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A STUDY OF ULCER OF THE STOMACH AND DUODENUM.

BASED UPON A SERIES OF EIGHTY-TWO CASES.

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(From the Clinic of Professor Osler.)

It is only by a careful study of a series of cases of any one disease that a general and comprehensive picture of the condition can be obtained. Moreover, an analysis of the cases of "round ulcer" at the Johns Hopkins Hospital is of particular interest, as the disease has here been regarded as comparatively rare.

Etiology. *Incidence.* In the analysis ulcer of the duodenum has been included, as physiologically, clinically, and pathologically the first part of the duodenum is identical with the stomach.

In all, 82 cases of round ulcer have occurred in the medical, surgical, and gynecological services from the opening of the hospital on May 5, 1889, to May 1, 1904—a period of about fifteen years. There has been a total number of 44,338 admissions to these services, which gives for the hospital a frequency of 0.18 per cent. According to service, the admissions are: 17,043 medical, with 76 cases; 16,553 surgical, with 4 cases; 10,961 gynecological, with 2 cases.

The surgical series includes only cases that were admitted primarily into that service, and is exclusive of the 11 that were transferred from the medical side for operation.

In this series there were six* duodenal ulcers—four in the medical and one each in the surgical and gynecological services.

* Since writing, the record of a subsequent admission of a supposed case has been found. The autopsy showed a ruptured œsophageal varix accompanying a cirrhosis of the liver to be the source of the hemorrhage. The local signs are difficult to explain, as no ulcer was found in the duodenum or stomach.

We have elsewhere* given comparative clinical data as to the frequency of ulcer (exclusive of duodenal) in some of the leading hospitals of Europe and America. We will recall some of the more striking figures: Edinburgh, 2.02 per cent.; Berlin, 1.33 per cent.; Boston, 1.28 per cent.; Montreal, 0.92 per cent.; London, 0.78 per cent.; New York, 0.44 per cent.; Baltimore, 0.37 per cent.; Denver, 0.12 per cent.

Hence we conclude that gastric ulcer is relatively rare in Baltimore.

SEX. The older writers state that ulcer of the stomach is more frequent in women, in the ratio of 2 to 1. Welch[1] found that in 1699 cases the ratio was 3 to 2. Mayo,[2] in the operating-room, found 59 per cent. in women and 41 per cent. in men. Greenough and Joslin[3] found in Boston a remarkably high ratio of females to males (5 to 1), "though no such disproportion exists in the total number of males and females admitted to the hospital." We could find only one higher ratio than the above, viz., 14 females to 1 male (quoted by Fiedler).

In our series there were 48 males to 28 females—a ratio of almost 2 to 1 in favor of the males. Now the ratio of total males to females for all services is 1 to 1, but 2 to 1 for the medical, from which the vast majority of our cases is taken. Hence it will be seen that gastric ulcer with us is *relatively* as common in men as in women, contrary to the usual idea.

Duodenal ulcer is more frequent in males. Osler[4] collected 178 males to 41 females—a ratio of more than 4 to 1. Mayo[2] found 20 males out of 24 cases at operation. In our series there were 5 males to 1 female—about the generally accepted proportion.

AGE. Most authorities, notably Welch in his classical monograph in Pepper's *System*, give the most frequent period for both sexes as between twenty to forty years; for the males, twenty to thirty years, and females, thirty to forty years. Greenough and Joslin's figures tally with this. Calwell,[5] in an analysis of 193 cases, found that in the male only 3 cases occurred before twenty-five years, while in the female there were no cases before thirteen, but the majority were between the ages of fifteen and twenty-five.

According to our table the larger number of all cases (32.9 per cent.) occurred between the ages of forty to fifty. In the males 41.5 per cent. occurred between the ages of forty to fifty—a decade later than usual; while in the females 44.8 per cent. occurred, as usual, in the third decade.

As one might expect, there were none in the first decade, and only 3 in the second, all of whom were girls. This rarity in early life is well recognized. Welch found only one instance of the disease in the first decade of life among the 607 cases which he col-

* Medical News, 1904, vol. ii.

lected. At the Baby's Hospital of New York there were only 5 cases in 390 autopsies. Fenwick,[6] in 1900, was able to collect from the literature only 18 cases in children. There were in our series 20 patients over fifty years of age—many of them coming under the category of "senile ulcer"—a condition often difficult to differentiate from carcinoma.

TABLE I.—SHOWING PROPORTION OF SEX AND AGE IN GASTRIC AND DUODENAL CASES.

Decades.	Total cases.	Per cent. of total.	Total males.	Per cent. of males.	Total females.	Per cent. of females.
1 to 9	0	0	0	0	0	0
10 " 19	3	3.6	0	0	3	10.3
20 " 29	17 (1 d.)	20.7	4	7.6	13 (1 d.)	44.8
30 " 39	15 (1 d.)	18.4	11 (1 d.)	20.7	4	13.9
40 " 49	27 (2 d.)	32.9	22 (2 d.)	41.5	5	17.2
50 " 59	14 (1 d.)	17.1	12 (1 d.)	22.6	2	6.9
60 " 69	5	6.1	3	5.7	2	6.9
70 " 79	1 (1 d.	1.2	1	1.9	0	0
	82	100.0	53	100.0	29	100.0

CONJUGAL CONDITION plays no role in the male. Of the 29 women, 16 of them were nulliparæ, 5 primiparæ, and 8 multiparæ.

RACE. Of the 82 cases, 67 were white and 15 were colored—a ratio of 4.5 to 1. As regards admission, the ratio is 7 to 1. Therefore, among our cases ulcer is relatively more common in the colored race, which fact does away with a recent suggestion that the rarity of ulcer in Baltimore is due to the number of colored admissions. We could find no other figures on this point.

NATIONALITY. Fifty-eight of our series were born in various parts of the United States—16 in Germany, 3 in Russia, 2 each in Scotland and Ireland, and 1 in Switzerland. The relative proportion of American-born to German-born patients is 13.6 to 1, while in our series it is 3.6 to 1—*i. e.*, ulcer is relatively more frequent among the Germans. This bears out our figures on the relative incidence of ulcer in America, which show that on this continent the percentage is 0.57, as compared with 0.78 per cent. of London, and 1.33 per cent. of Berlin.

OCCUPATION. Much stress was formerly laid upon occupation as a predisposing cause. Greenough and Joslin could not find any one occupation especially prone to the disease; thus 66 per cent. of the females were housewives, while of 200 consecutive female admissions there were 71 per cent.; again, only 7 per cent. were cooks as compared with 4 per cent. of general admissions. In our series 12 of 29 females did housework (41 per cent.), and only 3 were cooks (10 per cent.). Of 53 males there were 9 laborers and 9 merchants—*i. e.*, 17 per cent. of the cases were divided equally between the upper and lower walks of life. Bramwell believes that

ulcer is more common in the hospital class than in well-to-do patients —a point to which Robson rightly takes exception. Again, there were only 2 tailors and 1 shoemaker—occupations considered especially prone to the disease. The only part that occupation can play is that the in-door pursuit, on account of its sedentary nature, is more predisposing to ulcer than the out-door. Thus, in our series the proportion is nearly 4 to 1 (61 in-door to 14 out-door).

Family History. Heredity plays little or no part. In 8 cases (9.7 per cent.) there was a history of stomach trouble other than cancer, but in none was there any special suggestion of ulcer. In another 8 cases there was a definite history of carcinoma twice of the stomach (2.4 per cent.). Statistics elsewhere are unsatisfactory, though Drechsfeld succeeded in eliciting a family history of ulcer in 8 cases.

Previous Stomach Trouble. In 47 of the series (57.3 per cent.) there was a definite history of this for some months prior to the onset of symptoms suggestive of ulcer, though in many it was difficult to draw the line between the two. In the majority it was described as "indigestion," as shown by discomfort after certain articles of food, eructations of gas and pyrosis.

Chlorosis has been considered an important factor, especially among young females. Exact figures on this matter are difficult to obtain, though Osler states that "anæmia and chlorosis predispose strongly to gastric ulcer, particularly in women and in association with menstrual disorders." In 72 per cent. of Fenwick's series there was a definite history of chlorosis (pallor, œdema, and dyspnœa) before the first symptoms of ulcer were manifest. In only 9 of our cases (11 per cent.) was there a definite history of chlorosis.

Trauma. There was a positive history in 7 (8.5 per cent.). In 4 it was "direct violence," as a blow or fracture of ribs; in 1 "strain," and in 2 merely "injury." Burns may cause ulceration, but usually of the duodenum; of the latter there has not been an example in about 2300 autopsies at the Johns Hopkins Hospital.

Alcohol. In a large proportion (53.6 per cent.) there was a history of the use of alcohol, and in 19.5 per cent. to excess. How far this predisposes to ulcer it is difficult to say. Lancereaux thinks ulcer is more common in drunkards, especially those who drink ardent spirits. The weight of opinion regarding the "bodily ills" that are due to alcohol is at present turning the other way.

Syphilis. Syphilitic ulcer of the stomach is rare, and differs materially in its appearance and symptomatology from simple ulcer. We are not concerned with it in this paper. Nevertheless, in our series there was a definite history of syphilis—either recent or old—in 15.8 per cent Fenwick found such in 10 per cent. of the chronic cases. The etiological role that syphilis may play consists probably in the production of a specific endarteritis.

Tuberculosis. In our series, which does not include specific tuberculous ulcers, tuberculosis in one or another of its manifes-

tations was present in 13.4 per cent. of the cases, as shown post-mortem, or clinically by the sputum. Fenwick found such in 17 per cent. of his series There is no evidence in the literature that tuberculosis predisposes to ulcer. In Brinton's opinion, however, ulcer of the stomach predisposes to tuberculosis. Welch[1] says: "The large share taken by pulmonary phthisis in deaths from all causes renders the disease a frequent associate of gastric ulcer. It is *probable* that the lowered vitality of phthisical patients increases somewhat the liability to gastric ulcer."

Diseases of the Heart and Bloodvessels. Arteriosclerosis may play some part, as it predisposes to thrombosis, which is undoubtedly of importance in the pathogenesis of round ulcer. It was present, as shown clinically by palpation of the peripheral vessels or post-mortem by macroscopic changes, in 48.8 per cent. of our series; to a marked degree in 22 per cent.

Endocarditis should also be considered, as it may give rise to embolism, which has been regarded by some as an important etiological event. Valvular disease of the heart was present, as shown by undoubted physical signs or at autopsy, in 8.5 per cent. No regard is taken of merely hæmic murmurs which were noted in 5 other cases.

Symptomatology. *Complaints.* These were various, but in 50 per cent. "pain" was the chief complaint. In another, 47.5 per cent., "stomach trouble;" "dyspepsia" or "vomiting" were the symptoms from which they sought relief. In only 5 cases (6 per cent.) was a history of "vomiting of blood" volunteered by the patient.

Mode of Onset. There were only 8 cases (9.7 per cent.) that might be termed *acute*, of which two developed in the hospital during the course of another disease; 2 developed twenty-four hours before admission; 3 three weeks, and 1 four weeks before admission. The remaining 74 were examples of *chronic* "ulcer" having existed for periods ranging from two months to twenty years.

The Three Cardinal Symptoms. The order of frequency of these will be seen in Table II., where they are contrasted with those given by Greenough and Joslin,[3] and those of Fenwick. For comparison's sake we have also added those of Osler and McCrae[7] in their analysis of the carcinoma cases in this institution.

Table II.—Frequency of Vomiting, Pain, etc., in Gastric Ulcer.

Symptoms.	Johns Hopkins Hospital. (82 cases.)	Greenough and Joslin. (187 cases.)	Fenwick (chronic). (265 cases.)	Cancer series (J. H. H.) (150 cases.)
Vomiting . . .	85.3 per ct.	95.7 per ct.	72 per ct.	85.3 per ct.
Pain	82.9 "	92 5 "	100 "	86.6 "
Hæmatemesis . .	75 6 "	78.6 "	71 "	28.1 "
	Exclusive of melæna.	Exclusive of melæna.	Inclusive of melæna.	Exclusive of melæna.

By this it will be seen that the order of frequency in ours is similar to that of the Boston writers, though all three symptoms are somewhat more frequent in the latter. On the other hand, Fenwick's figures from London for 265 chronic cases are somewhat different, pain being present in 100 per cent.; while vomiting and hæmatemesis are of practically equal frequency, they are considerably less common than in either of the preceding series. The cancer figures are interesting, pain and vomiting being present in the vast majority, while hæmatemesis, as one might expect, is three times less frequent than in ulcer in our series.

PAIN. This symptom is usually the first indication of the existence of an ulcer, and is the most constant and distinctive feature. But pain is only a relative matter, and is the most difficult of all data to estimate. It was present in 82.9 per cent. of our cases. It usually means an active ulcer or its sequelæ. Leube believes that cicatrices cause pain only in very rare instances.

Site. Pain is usually referred to the epigastrium, as was the case in 54.4 per cent. of our series. Occasionally it is referred to the spine or left shoulder-blade (14.7 per cent.); more rarely to either hypochondrium (8.8 per cent.). In 2 cases the sternum and right iliac fossa were the respective locations. That the situation of the pain may give a clue to the site of the ulcer was maintained by Brinton. This is not accepted by the more recent authors. Of the 27 cases in which the site of the ulcer was determined at autopsy or exploratory operation, the site of pain was entirely misleading in 3 cases, of no definite value in 17, while in another 7 it only pointed to the pylorus or the duodenum.

Character. The pain is usually severe, though it may not exceed that of hyperchlorhydria. It is rarely continuous (4.8 per cent.), but if so usually means some complication as perigastric adhesions, localized peritonitis, or hyperchlorhydria. The *quality* of the pain is variously described as "sharp" (20.3 per cent.), "dull aching" (18.6 per cent.), "burning" (15.2 per cent.), "colicky" (13.5 per cent.), "gnawing or grinding" (11.9 per cent.), and as "boring," "cutting," "tearing," etc., in the remaining 22.5 per cent.

Pressure usually increases the pain, but may afford relief. The statements of different observers on this point are at great variance. In 24 cases where inquiry was made with regard to the question, increase of pain was present in 62.5 per cent., relief in 33.3 per cent., and no relief in 4.2 per cent. Again, pain is influenced by *posture.* Thus, out of 10 cases in which note is made of this, relief was experienced in 6 either by lateral or dorsal decubitus, "knee-and-chest" or upright position. Osborne's contention that the site of the ulcer may be inferred from the effect of posture on the pain, could only be tested in 2 cases, and without satisfactory result. In 1 case, where the ulcer involved the entire circumference of the pylorus,

dorsal decubitus gave relief, while in a duodenal ulcer with stenosis either lateral posture was helpful.

The relation of pain to *ingestion of food* is important. It is usually increased by eating (39 per cent.), but may be relieved (14 per cent.), or, again, be entirely independent of food. Gerhardt maintains that patients who are afraid to eat because they *fear* pain are usually suffering from simple ulcer and not cancer. There was "no pain" in only 4 cases, though in another 10 no mention is made. Some have called attention to the fact that cases of ulcer of the stomach without pain represent a grave type of the disease. In our series of four, 3 died, 2 from hemorrhage and 1 from a complication. In Boston 3 of 14 cases without pain were fatal.

Vomiting is the commonest symptom in our series (85.3 per cent.). There is a close relation between the severity of the pain and the vomiting. In the majority (67.2 per cent.) it is frequent, occurring almost constantly in a few cases; in others periodically. It is usually involuntary, but in some it may be provoked for the relief it affords, as was the case in 3 of our series. Again, in a certain number of cases (20.7 per cent.) it is only "only occasional," following some particular paroxysm of pain or the ingestion of certain foods. In a small percentage (12) it is the exception, and not the rule. The *quantity* varies greatly, but is profuse only where there is either hypersecretion or pyloric obstruction. Emesis generally depends upon the *ingestion of food*, and occurs at the height of digestion or immediately after taking food into the stomach. This was the case in 14 out of 20 where this point was noted It usually affords relief (as was the case in 15 of our series) by removing the cause of irritation, viz., the food and acid-juice. In 7 cases there was an entire absence of vomiting. These merit a moment's consideration; 5 ran a moderate course, 1 being discharged well, and 4 improved; the other 2 were more severe, 1 dying in the hospital, and 1 four weeks after discharge. The autopsy on the former (Case 15), revealed a large eroding ulcer upon the lesser curvature and posterior wall near the pylorus, with erosion of the artery, perforation of the wall, and exposure of the pancreas beneath. This one case conflicts with the statement "that vomiting is never absent when an orifice of the stomach is involved."

Relation of Vomiting and Pain to the Site of the Ulcer. Of the 23 cases that came to autopsy or exploratory operation, the site of the ulcer is specified in 22. In ulcer of the pylorus vomiting was almost constant, as was also pain, although to a somewhat less extent. In involvement of the lesser curvature vomiting and pain were present in a third of the cases only. In 4 cases in which the posterior wall was also involved, vomiting was present in 3 and pain in all 4. In the 1 case, in which the anterior wall was also involved, both were present. In 4 cases with ulcer on the greater curvature involving either wall pain was present in three instances,

vomiting in all. In 1 of the 2 cases in which the lesion was on the posterior wall alone, both were present. Thus, vomiting was present in 17 out of 22 cases, more constantly where either orifice or the greater curvature was involved than when the ulcer was on the lesser curvature. Pain, on the other hand, was practically constant except in ulcer of the pylorus or lesser curvature.

TABLE III.—RELATION OF VOMITING AND PAIN TO SITE OF ULCER.

Site of ulcer.	No. of cases.	Vomiting.	Pain.
Pylorus	8	7	5
Lesser curvature	3	1	1
Lesser curvature and posterior wall	4	3	4
Lesser curvature and anterior wall	1	1	1
Greater curvature (near cardia)	1	1	1
Greater curvature and posterior wall	1	1	...
Greater curvature and anterior wall	2	2	2
Posterior wall	2	1	1
	22	17	15

HÆMATEMESIS. Vomiting of blood is usually not so constant a symptom. It was, however, present in 75.6 per cent. of our series, as compared with 78.6 per cent. of the Boston cases. The older writers give a much lower frequency. Thus, Brinton, 28 per cent.; Müller, 32 per cent., and Gerhardt, 47 per cent.

The *color* is noted in 56 cases; "coffee-ground" in 66 per cent.; bright red in 33 per cent. "Coffee-ground" vomiting occurs whenever the blood remains sufficiently long in the stomach to be acted upon by the gastric juice, and does not necessarily imply malignancy. On the other hand, "bright blood" was present in 11 per cent. of the cancer series in which hæmatemesis occurred. The *quantity* was usually fairly large; in 86.1 per cent. it was more, and in 18.9 per cent. it was less than two drachms. Severe hemorrhage may cause instant death without external evidence. This "l'hémorragie foudroyante" occurs, according to Brinton, in 5 per cent., and Fenwick in 3.4 per cent. There was one example of this in our series (Case 65), in which an aneurysm of the thoracic aorta being present, its rupture was thought to be the source of hemorrhage, until the autopsy revealed an ulcer eroding the gastric artery. The *number* of the hemorrhages is also variable. Thus there were—

One hemorrhage	7 cases.
Two hemorrhages	7 "
Three hemorrhages	11 "
Four hemorrhages	1 case.
Many hemorrhages	15 cases.

In about 40 per cent. of Fenwick's cases it recurred in a week, in 73 per cent. in twelve months after its apparent cessation. The factor which determines the recurrence, Moynihan[8] thinks, is the stretching of the ulcer from distention of the stomach. This latter condition he found in all of 15 ulcer cases in which he operated

for hemorrhage. Hæmatemesis sometimes gives almost immediate relief to the pain and vomiting. Of the 16 cases in which no hæmatemesis occurred ulcer was found in 5 at autopsy and 1 at operation; in the remaining 10 the diagnosis was supported by other data, such as melæna, local tenderness, vomiting, etc. As to the frequency of hæmatemesis at the menstrual period we have no data. In two of the Boston series the first hæmatemesis occurred at this time.

MELÆNA. Severe hemorrhage from an ulcer invariably gives rise to tarry stools, but, according to Hosslin, only when the blood amounts to 500 c.c. Melæna can occur without hæmatemesis in an ulcer near the pylorus or in the duodenum. In 55 cases in which there was a clear history as to its occurrence or non-occurrence melæna occurred in 80 per cent. The statements of patients are not reliable on this point, and often give one an altogether erroneous idea. There is no doubt that a careful, systematic, routine examination of the stools would reveal blood in the vast majority of cases.

The *color* was dark and tarry in 8 per cent., and bright red in 20 per cent. of 28 cases. The color varies, of course, according to the size, freshness, and rapidity of the hemorrhage and the length of time the blood takes to pass through the intestinal tract. The *quantity* was large in 9, small in 6. The *number* of times in which melæna was observed varied from a few in 17 to many in 10.

Of the 44 positive cases, 36 accompanied or followed the vomiting of blood; 8 gave no history of hæmatemesis; 1 of these was regarded as duodenal, and 5 were considered gastric ulcer; 2 were shown at autopsy to be gastric ulcer.

NAUSEA was present in 32 out of 34 cases. That this symptom was not noted in the remaining 50 cases suggests that it is relatively uncommon. This is natural, for the vomiting is a result of the pain and a desire on the part of the patient or his stomach to be rid of irritating material. A recent writer[9] says: "A feature to be remembered about the vomiting is that it is not usually accompanied by nausea, and does not require much effort."

DYSPEPSIA. By this term we include discomfort after eating, eructations, pyrosis, headache, palpitation, meteorism, etc. It was present in 96 per cent. of 52 cases. It is of little or no importance, and generally dependent upon the attendant hyperchlorhydria and chronic gastritis, both of which usually accompany the disease. Digestion, then, is not, as a rule, more difficult or restricted in uncomplicated cases.

APPETITE. This may be lost (5.9 per cent.), variable (4.4 per cent.), poor (45.6 per cent.), or good (44.1 per cent.). Others have found that as a general rule the appetite is good. The dictum that in ulcer the appetite is good, but patients *fear* to eat, while in cancer the appetite is lost, is an important aid to diagnosis.

BOWELS. Constipation is the rule, as was the case in 61.6 per cent. here and 80 per cent. in Boston. Greenough and Joslin ascribe their large percentage to the greater frequency of the disease in women. The latter was not the case in our series, which may account for the lower percentage in which constipation was present. The most important cause is the small amount of solid food taken and retained by the patient. In 10.9 per cent. there was "looseness" if not actual diarrhœa. In the remaining 27.5 per cent. the bowels were regular.

GENERAL HEALTH. The usual teaching that moderate loss of weight and strength are present is not borne out. In none was cachexia noted, but in a very large number was there loss of strength (98 per cent. of 47 cases) and loss of weight (87 per cent. of 62 cases) noted. These are due partly to the pain and inability to eat, and partly to the loss of blood. I think the number of cases (87.1 per cent.) in which loss of weight occurred is of interest. The details are given in Table IV.

Loss of weight (no figures)	15		
Slight (1–5 lbs.)	1	10–20 lbs.	10
Moderate (5–10 lbs.)	2	20–30 lbs.	7
Marked (10 lbs. or more)	36	30–40 lbs.	10
	54	40 + lbs.	9
			36

Of the 9 that lost forty pounds or over, there were 3 that lost forty pounds; 2, forty to fifty pounds; 2, forty-six pounds; 1, fifty pounds, and 1, seventy-five pounds. Such a marked loss of weight would naturally suggest malignant disease, but it must be borne in mind that pain, coffee-ground vomitus, tumor, and marked loss in weight can all be present in benign ulcer of the stomach.

PHYSICAL SIGNS. *Tenderness* is the least important of the cardinal symptoms or signs. Its absence or presence is responsible for many errors in diagnosis. It was noted in 57.9 per cent. of our series as compared with 69.5 per cent. in Boston. The *degree* varied from one extreme to another; thus, 2 cases could not bear the pressure of the clothing, while in others considerable pressure was required to elicit any discomfort.

Site. It was usually localized to a small area in the epigastrium (75 per cent.), but in others it was general over the upper abdominal zone. It was present in the left hypochondrium (10 per cent.); in the right hypochondrium (5 per cent.); right iliac fossa (5 per cent.), and in umbilical zone (5 per cent.). The *tenderness in back* (Druckschmerz), which, according to Boas, is present in 30 per cent., has not been sufficiently tested in this hospital to warrant any conclusions.

PRESENCE OF RESISTANCE OR MASS. Out of 70 cases where this point is noted a mass was present in 20 per cent. and resistance in 14.2 per cent. The mass was in the right hypochondrium (36 per

cent.), epigastrium (29 per cent.), greater curvature (14 per cent.), pylorus (14 per cent.), and left hypochondrium (7 per cent.). The presence of a mass is often misleading, but the possibility of such, while not so frequent as in carcinoma, should always be kept in mind. In Osler and McCrae's[7] series of cases it was noted in 76.6 per cent.—only about three and one-half times more frequent than in our series. Reinhard has reported 16 cases with tumor in which simple ulcer was found at autopsy. The Mayos[2] have seen at operation 6 cases with tumor due to thickening about a chronic ulcer. Moynihan[8] in 100 gastroenterostomies for ulcer, found 10 cases with a tumor, 2 of which were thought to be malignant until the exploratory operation and subsequent course pointed to their benign nature. Gerhardt has described four conditions in which a mass may be present in a simple ulcer, viz.: 1. The thickened base and hard margin of the ulcer itself. 2. Functional hypertrophy of the musculature. 3. Localized exudate or abscess from perforation. 4. Adhesions between neighboring organs. There were examples of the three latter in our series. One case has been reported in detail by Osler[10] (Case 12) on account of the "prominent tumor at the pylorus, which relaxes and contracts and appears and disappears beneath the skin, due to the hypertrophied pyloric musculature."

Dilatation of the Stomach. This was present clinically in 18 out of 52 cases (34.6 per cent.); an absence of comment in the remaining 30 cases would warrant assuming the absence of dilatation. Therefore, a percentage of 22 would seem a fairer figure. As to the *degree*, it was marked in 7, moderate in 5, slight in 2, and not specified in 4. In the cancer series it was present in 42 out of 150 cases (21.3 per cent.). Broadbent has pointed out that extreme dilatation from carcinoma is rare. Osler and McCrae refute this, as they found a much larger number of cases of dilatation of the stomach due to malignant disease than any other single cause. Our figures place simple ulcer as *relatively* as frequent a cause, but on account of the greater prevalence of cancer in the Johns Hopkins Hospital, malignant disease would still take pre-eminence as a cause of gastrectasis. Dilatation in an ulcer signifies pyloric obstruction either directly from the ulcer, or scar, or perigastric adhesions, or indirectly from contraction or spasm of the pylorus from mechanical irritation. It must be remembered that there may be mechanical obstruction from contraction or spasm caused by a pyloric ulcer, *without* dilatation of the stomach (Hartmann's syndrome). The dilatation was determined by the tartaric acid and soda bicarbonate method *without* any harmful results. Hockman, however, has reported deaths from sudden distention of the stomach.

Gastric Analysis. The value of an elaborate analysis of the gastric juice is much exaggerated. We quite agree with Mayo that

the hydrochloric acid determination is the only one of value, and would add that *too much* importance cannot be attached to it. The Ewald-Boas breakfast is the one in vogue in this institution as elsewhere in America. The objection that exists in England to the use of the stomach tube in ulcer of the stomach does not prevail here. Unless there was a recent history of hemorrhage a test breakfast was given, and without any ill effects. To 53 patients a breakfast was given, and in only 1 case was a Riegel meal employed. Only 1 patient refused to submit. Of the 54 cases, no contents were recovered in 2. Analysis from one or more breakfasts was made in 52 patients; thus, 29 had one meal, 14 two meals, 5 three meals, 3 four meals, 1 six meals. Let us examine the results in detail.

Total Amount. Assuming that 40 to 50 c.c. be normal residue in one hour for the Ewald-Boas breakfast, and 300 to 350 c.c. at the end of two to three hours for the Riegel meal, there were 46 cases in which no figures were given, 54 per cent. above and 17 per cent. below normal, while the remaining 29 per cent. were within the limits of normal. It is interesting to note that many cases of Reichman's disease, where hypersecretion exists to an extreme degree, have been recently shown to be due to old cicatricial stenosis of the pylorus or duodenum.

Total Acidity. If we assume that normally after an Ewald test breakfast from 40 to 60 c.c. of decinormal sodium hydrate solution are required to neutralize 100 c.c. of filtered gastric contents, 27.5 per cent. of the cases showed hyperacidity, 42.5 subacidity, while the remaining 30 per cent. were within normal limits.

Free Hydrochloric Acid. This was tested qualitatively by Günzburg's reagent, and was found present in 82 per cent., and absent in only 18 per cent. of the cases. In 34 cases the quantity was estimated by tritration with dimethyl-amido-azo-benzol as indicator, which is undoubtedly less accurate than tropaolin. There was hyperchlorhydria in only 17.6 per cent.; the quantity of HCl was within normal limits in 26.4 per cent.; there was hypochlorhydria in 26.4 per cent.; in 1 case the free HCl was normal once, and below normal on the next examination. This is, however, in marked contrast with the results in our cancer series, where free HCl was absent in 92 per cent. These figures are rather contrary to the generally accepted statement that hyperchlorhydria is present in 75 to 80 per cent. of cases, as is held by Riegel and many others. Mayo[2] in a recent paper says "two facts are prominent, first, that 75 per cent. of gastric ulcers lie in the grinding apparatus of the pyloric end, and, second, that sometime in the history of gastric ulcer, and in the majority of cases at all times, there is a relative increase in the secretion of HCl, and, whether cause or effect, this excess is a most important etiological factor."

We repeat, our figures do not bear this out, nor have Ewald, Gerhardt, or Drechsfeld found such to be the case.

Lactic Acid. This was tested for in 43 of the cases, and was found present in 6 (14 per cent.), doubtful in 3 (7 per cent.), and negative in 34 (79 per cent.). In these 9 cases there was dilatation of the stomach in 4, subacidity without evident dilatation in 2, and neither dilatation nor subacidity in 3. In every case Uffelmann's reagent was used, but in only 2 was the juice previously extracted with ether. Let us glance at a brief summary of these 9 cases.

(*a*) *Positive Cases.* 1. Case 6.[28] Pyloric stenosis (cicatrix of ulcer); female, aged twenty-two years, colored, washerwoman; hospital No. 5646; admitted July 29, 1892. Absence of previous dyspepsia; sharp onset of symptoms of pyloric stenosis; marked emaciation; palpable tumor; dilatation of stomach with stagnation; test for lactic acid very positive on four occasions; that on August 22d, after daily lavage, showed lactic acid with absence of free hydrochloric. Operation; death; autopsy.

This case has been criticised by Boas as probably one of ulcus carcinomatosum. There can be no ground for this objection, as a careful gross and microscopic examination of the tissues was made. Dr. Thayer tells me, however, that the stomach was not washed out before each test breakfast as a preliminary precaution.

2. Case 15. Ulcer of the stomach; perforation with local peritonitis; hemorrhage. Male, aged forty-five years, piano-tuner; hospital No. 9473; admitted March 22, 1894. History of dyspepsia for six months, vomiting, but no hæmatemesis; marked loss of weight. No dilatation of stomach. Analysis of test breakfast on 23d instant showed no free HCl and no lactic acid, but vomitus of same date contained a faint trace. Readmitted on June 16, 1894, with a recent history of hæmatemesis. Test breakfast without previous lavage showed stagnation; faint HCl, and presence of lactic acid. Diagnosis on both admissions: cancer of stomach. Readmitted September 11, 1894, in a very grave condition; sudden death; autopsy.

3. Case 19. Gastric ulcer. Male, aged forty-eight years, cabinetmaker; hospital No. 11,250; admitted October 27, 1894. History of ulcer for four years. No dilatation of stomach. Test meals on the 29th and 30th show presence of HCl and also of lactic acid; latter disappeared two days later, though there was no note of gastric lavage; slight stagnation.

4. Case 34. Gastric ulcer. Male, aged forty-eight years; traveller; hospital No. 17,089. Admitted August 24, 1896. History of ulcer for twelve years. Test meal and specimen of vomitus give very slight reaction to free HCl and lactic acid. Stagnation slight. No note of previous lavage and no dilatation.

5. Case 36. Gastric ulcer. Male, aged forty-five years, laborer; hospital No. 18,427; admitted January 27, 1896. Ulcer history for two years; slight dilatation of stomach; only one test meal given,

which showed free HCl and slight reaction for lactic acid. Stagnation evident. No lavage. Discharged well.

6. Case 56. Cirrhosis of liver; cholelithiasis; ulcer of stomach. Female, aged forty-two years, laundress; hospital No. 31,023. Admitted June 27, 1900. History of cirrhosis of liver and alcoholic neuritis. No definite history of ulcer. Two test meals *after lavage* on previous night showed presence of blood and a definite reaction for lactic acid in ethereal extract. Readmitted on September 17, 1900, deeply jaundiced and comatose. No test meal. Death; autopsy.

(*b*) *Doubtful Cases.* 1. Case 10. Gastric ulcer. Male, aged thirty-seven years, oysterman; hospital No. 7743. Admitted July 20, 1893. History of ulcer for eight years; test breakfast on 21st showed free HCl and no lactic acid, but on the 25th instant good free HCl and a small amount of lactic acid. No note of lavage or dilatation. Discharged.

2. Case 30. Gastric ulcer. Aged thirty years, teacher; hospital No. 14,899. Admitted July 4, 1896. Ulcer history for two years; test meal on the 7th showed free HCl and a faint lactic acid reaction. No lavage and no dilatation. Discharged.

3. Case 54. Pyloric stenosis. Dilatation of stomach. Male, aged thirty-eight years, warehouseman; hospital No. 28,202. Admitted October 21, 1899. History of ulcer for nine months and stenosis for four; after previous lavage, on the 23d, there was no free HCl and a "suspicion of lactic acid," but on the 27th free HCl was found and no lactic acid. In spite of daily lavage, on November 7th and again on December 7th there was noted a suspicion of lactic acid and absence of free HCl. Operation; death; autopsy.

Hence it will be seen that in spite of gastric lavage before the test breakfast lactic acid was present in 2 cases—once on six occasions and once in small quantities on three occasions. In 3 of the cases daily lavage was performed without a disappearance of the lactic acid. In the other 4 cases there was no lavage instituted either before the meal or at regular intervals. We believe that the presence of the acid in these cases is due to stagnation of the gastric contents and a consequent fermentation, and in those 2 cases which still showed lactic acid, though after the prolonged use of lavage, the stagnation of even twenty-four hours was sufficient to account for the acid. Unfortunately we have no case illustrating the disappearance of the lactic acid with the relief of the stagnation by a more frequent use of the stomach tube. Lactic acid was present in 75.3 per cent. of the cancer series as contrasted with 14 per cent of our cases.

Blood was noted macroscopically in 18 out of 33 cases—in large amounts in 2, in small amounts in 7, in traces only in 2. In another 5 it was found microscopically alone. Thus, blood was present in 57.5 per cent. as compared with 26.6 per cent. of the cancer series.

Microscopic examination was recorded in 33 cases. It was negative in 18 instances. Red blood corpuscles were found in 8, red blood corpuscles and "bacteria" in 3, and bacilli in 4. The latter were in each case thought to be Oppler-Boas bacilli.

THE BLOOD. The alterations in the blood are those seen in any secondary anæmia due to hemorrhage—*i. e.*, of the chlorotic type. Rencki,[11] in 1901, after a careful study of the blood in 15 cases of cancer and 14 of ulcer, concluded that no characteristic differences could be made out between the anæmias of the two, and that examination of the blood is of very little value from a diagnostic point of view.

Red Blood Corpuscles. The red blood corpuscles were counted in 44 of our cases. The highest count was 6,784,000, the lowest 1,012,000, and the average of sixty-seven counts was 4,071,000 per c.mm. The highest count occurred in a patient (Case 67), who had almost constant vomiting, for which she was on rectal feeding for some time; the resulting concentration of the blood was the probable cause of the polycythæmia. The lowest count was in a case (Case 15) which was regarded on two previous admissions as cancer; indeed, it was not until the autopsy after the third admission that the true nature of the case was discovered. Where anæmia occurred it was due to hæmatemesis. In 1 case there was a difference of 4,332,000 per c.mm. between the count of the first and that of the third admission after a series of hemorrhages. There were 10 cases in which the red corpuscles were below 3,000,000, and 2 in which they were between 3,000,000 and 3,500,000 per c.mm. Osler quotes a case of duodenal ulcer with hemorrhage in which the red cells fell to 700,000 per c.mm. The average red count in Cabot's[12] series (51) was 3,372,000 per c.mm.; he states that "there is no single disease in which the red blood cells are apt to be so low, with the exception of pernicious anæmia." Greenough and Joslin report counts in 43 of their series, and in only 24 was the red count below 4,000,000 per c.mm. Grawitz suggests that the ulcerative process itself, *without* hemorrhage, may cause actual anæmia, but Cabot believes that it is uncommon to see marked anæmia in patients who have never had hæmatemesis.

White Blood Corpuscles. These were counted in 54 cases. The highest was 40,000, the lowest 1100 per c.mm.; the average of sixty-three counts was 7500 per c.mm. The leukocytosis of 40,000 (Case 15) is difficult to explain, unless it was due to the recent hemorrhage. Leukocytosis does occur under these circumstances, though usually to a less marked degree. Unfortunately there were no differential counts in our series. Digestive leukocytosis was tested for in 5 cases and was present in all, though never to a marked extent. The highest count was 10,500 per c.mm. The digestive leukocytosis is of very doubtful value in differentiating ulcer from cancer of the stomach, as has been pointed out by Osler and

McCrae, and later by Rencki. The former found it present in 10 and absent in 12 of their cancer series.

Hæmoglobin. This was estimated in 44 cases. The highest was 105 per cent., the lowest 12 per cent.; the average of sixty-two estimations was 58 per cent. The greatest variation in any one admission (Case 15) was 36 per cent. Cabot found the hæmoglobin reduced to a point below 50 per cent. in 80 per cent. of his cases. The average color index was 0.72, as compared with 0.67 of Greenough and Joslin. In the cancer series the average percentage of hæmoglobin was lower, being 49.9 per cent., while the color-index was 0.63. Hence the blood picture is that of a secondary or chlorotic anæmia, as seen from the following average determinations: red blood corpuscles, 4,071,000; white blood corpuscles, 7500; hæmoglobin, 58 per cent.

URINE. The pathological changes in the urine are not striking, and are usually accidental. Quincke says the urine is pale, abundant, and sometimes contains albumin. Albumin and casts were present in 15 of our series; a trace of albumin in 7. In 55 the urine was negative. In no case was there a quantitative estimation of the chlorides. Drechsfeld says the chlorides are often diminished and the phosphates increased. If dilatation be marked and fermentation increased there may be an excess of ethereal salts and indican. Acetone and diacetic acid were not noted.

FEVER. This was present in 30 per cent. of our series. It did not exceed a degree or a degree and a half except in 6 cases. In 2 of these it was continuous, the ulcer being a complication of typhoid fever and pneumococcic septicæmia, respectively. In 4 other cases there was irregular fever, associated in three instances with complications—general peritonitis, tuberculosis, pyloric stenosis.

COMPLICATIONS. *Pyloric Obstruction.* Of the 76 cases of gastric ulcer there were 5 with definite pyloric obstruction and 1 doubtful case; 4 of these were confirmed at autopsy. There were also 2 cases of duodenal stenosis, both of which were confirmed at autopsy or operation. This gives 8 cases (9.7 per cent.) of obstruction from ulcer or its scar. There were in addition 10 other cases in which clinically the stomach was dilated, but no definite obstruction to its outlet was thought to exist. At autopsy the stomach was found dilated in 5 of these.

Deaths from Hemorrhage. Hemorrhage was the cause of death in 7 cases (8.5 per cent.), 1 of which was a duodenal ulcer. Of these, 4 were males and 3 females—*i. e.*, there was fatal hemorrhage from gastric ulcer in 4 out of 48 males (8.3 per cent.), and 2 out of 28 females (7.1 per cent.), the third case above mentioned having been one of duodenal ulcer. Our figures bear out the usual statement that hemorrhage is more fatal in the male than in the female. In the Boston series there were 5 males to 2 females. The ages of the males were forty-three, forty-five, forty-nine, and sixty-six, or an

average of 50.7 years; the ages of the females were ten and fifty, an average of thirty years. The case in which fatal hemorrhage occurred from a duodenal ulcer was in a girl, aged twenty-one years. That hemorrhage is relatively more fatal in the young female cannot be deduced from 2 cases. Hood, of London, in a period of twenty years at Guy's Hospital, found no case of fatal hemorrahge in a young woman. The latter is also supported by the Boston writers.

In all 7 cases the bleeding point was found at autopsy. In 2 other cases of gastric ulcer and 1 other of duodenal there was a profuse terminal hemorrhage with the gastrointestinal tract filled with blood, but no sign of a bleeding vessel was found.

Opinion as to the frequency of fatal hemorrhage is at great variance. Mayo Robson[13] says it is a very serious and common accident occurring in 7 per cent. Byrom Bramwell,[14] in his controversy with Robson on this point, stated that he had only 2 cases in his hospital or private practice in a series of 127 cases (1.57 per cent.), and was able to find only an incidence of 0.67 per cent. in the Edinburgh Royal Infirmary and 0.84 per cent. in St. Thomas' (London). Welch says it is the cause of death in from 3 to 5 per cent. The other authorities in numerical order are: Müller, 11 per cent.; Rodman, 8 per cent.; Steiner, 6.3 per cent.; Brinton and Debove, 5 per cent.; Drechsfeld, 3.5 per cent.; Lebert, 3 per cent.; Schulz, 1.7 per cent. The fatal nature of the hemorrhage depends upon the fact that the ulcer makes a lateral opening into the vessel, which prevents natural hæmostasis by the retraction and contraction of the artery. It is impossible to correctly estimate the size of the perforated vessel from the amount of blood or the length of survival of the patient. Robson[13] states that "capillary hemorrhage may be so free as to simulate rupture of a large vessel, yet after death a careful examination may fail to discover any gross vascular lesion. Dieulafoy has pointed out that severe hemorrhage may occur as the result of a "pore-like erosion" of the wall of one of the large branches of the gastric arteries without any marked ulceration of the surrounding mucous membrane, or, at most, accompanied by an abrasion so slight in degree that even at post-mortem the lesion may be overlooked (exulceratio simplex).

Hemorrhage may be capillary, venous, or arterial. The latter is the most common. Thus, Savariand, in a series of 54 cases, found ulceration of the splenic artery in 17, of the coronary in 6, pancreaticoduodenal, 7; gastric arterioles, 10; branches of the coronary vein, 2; other veins, 2; vessel not determined, 10.

In our series hemorrhage was from an erosion of a vessel in 6 gastric ulcers and 1 duodenal. In Case 15 the splenic artery was eroded, in another the arteria corona dextra, while in the remaining 5 it was a small arteriole or vein.

As to the *percentage of deaths* in gastric ulcer that are due to

hemorrhage, Bramwell gives 9.3 per cent.; Fenwick, 16 per cent.; Cordui, 20 per cent., and Welch, 27 per cent.

Exclusive of the 3 cases in which no bleeding point could be found, and in which hemorrhage was not absolutely proven to be the cause of death, there were 7 out of 24 deaths (29.2) due to hemorrhage.

PERFORATION. (*a*) *With General Peritonitis.* Only 1 case occurred in our series of 82 ulcers (1.2 per cent.), which is a remarkable fact when one considers that there have been somewhat more than 47,000 admissions in the past fifteen years to all services. The history of this case in brief is:

Case 40. Male, aged fifty-eight years, colored; hospital No. 19,664; admitted June 8, 1897, complaining of pain and vomiting; gastric symptoms for one year; acute onset four days before admission, with pain in upper abdomen and vomiting. No hæmatemesis and no melæna; great weakness and emaciation; collapse; signs of general peritonitis on admission; temperature, 100° to 103°; pulse, 120 to 132; respirations, 36 to 48. The case was considered inoperable by the surgeon, but after twelve hours there was some improvement and an exploratory laparotomy was performed. General peritonitis was present, and a small ulcer on the anterior wall of the pylorus was found, through which the gastric contents were exuding; the patient's condition was so bad that nothing further could be done, and death ensued shortly after the patient left the table. Only a partial autopsy was permitted, which revealed in addition to the perforated ulcer a small scar in its neighborhood.

(*b*) *With Localized Peritonitis.* There are two examples of this: one (Case 15) showed perforation through the posterior wall with localized abscess between the pancreas and the left lobe of the liver, evidently a gradual process of considerable duration. The second (Case 65) showed perforation of the posterior wall into the pancreas, which was considerably eroded and firmly adherent to the stomach. Including these 2 cases there was perforation of the ulcer in 3 out of 82 cases (3.6 per cent.), in 3 out of 24 deaths (12.4 per cent.). There was no case of perforation of a duodenal ulcer.

Greenough and Joslin found perforation in 3.2 per cent. in Boston. Pathological statistics exaggerate the frequency of perforation. The only trustworthy method is to observe the number of times that perforation occurred in a large series of clinical cases. Lebert found 3.5 per cent. of 252 cases; Fenwick, in 5.5 per cent. of 265 cases. Brunner has recently analyzed 380 gastric and 86 duodenal perforations, which have been reported between 1893 and 1903. He found that the accident occurs in about 2 to 4 per cent. of cases; it is more common in the gastric ulcer in England, and the duodenal in America. Only one instance was on record of perforation while the patient was at rest in bed under medical treatment; in another case it followed gastroenterostomy. Perforation is possible

in any situation in which ulcer may form, but is more frequent in the anterior wall near the pylorus and lesser curvature, or in the anterior wall of the upper horizontal portion of the duodenum. There is a certain small number of cases (7.5 per cent. of gastric and 20 per cent. of duodenal) in which perforation occurs without a previous history of ulcer. Brunner considers a diminution in liver dulness unassociated with tympanites characteristic of perforation of an ulcer; also that the site of pain is usually localized to the left of the median line in gastric and to the right in duodenal ulcer, and, further, that the area of tenderness in duodenal ulcer is not in the locality of the pain, but in the right iliac fossa.

Recovery from acute general peritonitis following perforation of a gastric ulcer may occur spontaneously. There are eight examples in literature.

Parotitis occurred in 1 case (1.2 per cent.), which has been reported in detail by Dr. A. Duval Atkinson.[17] A brief summary is as follows:

Case 29. L. P., female, aged thirty-five years; hospital No. 14,749; housemaid; admitted December 16, 1895, complaining of vertigo and general weakness. Family and personal history negative. For the past year has not been well, having continual pain in epigastrium, for one month, and amenorrhœa, for four months. On December 9th she took to bed because of gnawing sensation in epigastrium and nausea, and moderate hæmatemesis; latter was repeated in four hours, and was followed by extreme weakness. Examination on admission showed pallor of skin and mucous membranes; slight increase in cardiac dulness and a mitral systolic murmur; abdomen negative. Red blood corpuscles, 2,214,000; white blood corpuscles, 8000; hæmoglobin, 30 per cent. Urine had a specific gravity of 1010 to 1006, trace of albumin, and granular and hyaline casts. On December 16th there were pain and swelling of the left parotid, which by the next day had extended above the zygoma and far behind the mastoid, with infiltration of the side of the neck; locally redness, heat, and tenseness. On incision creamy pus exuded, which in cover-slips and cultures showed streptococci. No change in local condition, but patient gradually sank, and died on the 24th instant. There was no autopsy permitted.

This is not a very rare complication. Thus, Stephen Paget[18] reported 2 cases in 1897, and Hone[19] in the same year collected 8 cases from the literature and added 1 of his own. The latter was in a female, aged thirty-nine years, with a history of gastric ulcer for six years, with repeated hæmatemesis, who developed a double parotitis with little suppuration. Death; no autopsy permitted. This condition in ulcer is analogous to that found in injuries and diseases of the peritoneum, generative and abdominal organs. Some consider it more frequent in cases on rectal alimentation,

which is denied by Hone, who considers that hæmatemesis is a predisposing factor.

Tetany. We have only one illustration of this, which, though not recognized at the time, was probably tetany. It occurred in Case 29, reported above. This patient gave a history of "cramps" in the legs for one year, of the utmost severity, being of a shooting, darting character, coming on in paroxysms, and lasting several minutes. At the height of these pains a feeling of "pins and needles" would be felt in the hands and fingers. There was no evident gastric dilatation. It must be borne in mind that the milder grades of tetany may be overlooked entirely, such as muscular cramps and pricking in the extremities without typical contractures. When severe, tetany usually terminates fatally. Thus, Hochnard reported 10 deaths in 11 cases and Albu 31 out of 40.

Ulcus Carcinomatosum. There are 3 cases in this series in which cancer was supposed to have developed upon a pre-existing ulcer. Two of these (1 and 2) are very doubtful, and though diagnosed by the resident physician "carcinoma of the stomach (post-ulco)," they were considered merely cancer by Dr. Osler in his series. The history of these cases in detail is:

1. Case 26. Female, aged sixty years; hospital No. 12,287; admitted March 19, 1895, complaining of indigestion. For past year abdominal pain, worse after food and at night; vomiting of small amounts; hæmatemesis for two or three days one year ago; occasionally melæna; appetite good; bowels constipated; loss of weight. No tenderness, but mass to right of umbilicus; marked dilatation of stomach; analysis showed free HCl and total acidity of 77; discharged much improved.

2. Case 39. Male, aged forty-seven years, German, shoemaker; hospital No. 18,818; admitted March 6, 1897, complaining of "indigestion" for six years, and "vomiting," burning pain in perigastrium after meals, relieved by "knee-chest" posture; vomiting for four months, at first slight, now frequent, and large in amount. One attack of hæmatemesis four to five months ago, when on four or five occasions he vomited dark lumps of blood; loss of thirty-three pounds in weight; no tenderness; no mass; marked dilatation of stomach. Analysis gave a trace of free HCl and no lactic acid; hæmoglobin, 65 per cent.; red blood corpuscles, 6,120,000; white blood corpuscles, 8000. Patient discharged improved.

3. Case 64. Male, aged fifty-five years, colored, oyster shucker; hospital No. 33,849; admitted February 13, 1901. Patient was unconscious, and the history elicited from his wife did not point to the stomach. He died from a pneumococcus septicæmia, and at autopsy an old round ulcer was found on the lesser curvature of the stomach 7 cm. from the pylorus. Microscopic sections showed "in the depth of tissues glandular structures, in which epithelial cells are actually invading the surrounding tissue. This invasion is also to be seen

in the mucosa immediately adjacent to the ulcer." At first a diagnosis of "gastric ulcer with beginning carcinomatous change" was made, but Dr. McCallum, the resident pathologist, on re-examining the sections thought "that the slight new-growth of epithelium present is not more than might be accounted for in the regeneration process in the healing of the ulcer." There were no metastases or invasion of the surrounding tissue.

4. (This case was not included in our series, through an oversight.) Male, aged forty-nine years, painter; hospital No. 44,935; admitted December 7, 1903, for "stomach trouble." Past history negative except for syphilis in 1880. Present illness began three years ago with "heartburn" and acid eructation. In June, 1901, a profuse hæmatemesis from which he fainted, followed by tarry stools; frequent vomiting since onset, often very profuse; appetite good until nine months ago, when he gradually gave up solid food because of pain; loss of fifteen pounds in weight during the past five weeks; no emaciation or cachexia. Local tenderness in epigastrium; active visible peristalsis and marked dilatation of stomach; firm, nodular mass to right of mid-line and 3 cm. above umbilicus. Hæmoglobin, 45 per cent.; red blood corpuscles, 3,004,000; white blood corpuscles, 15,200. Test breakfast showed marked hypersecretion and hyperchlorhydria (100.5), with blood-streaked mucus. The hyperchlorhydria practically decreased with gastric lavage, until by December 18th there was a marked subacidity. There was always a morning residue of 400 to 2000 c.c. The general condition improved; there was a gain of two pounds in weight, and the blood rose to 60 per cent. hæmoglobin and 4,000,000 red blood corpuscles. The patient was transferred to the surgical side, and an operation was performed December 30, 1903. At the exploratory operation there was marked dilatation of the stomach, with a mass surrounding the upper two-thirds of the pylorus. Several large, hard glands were found, one of which was very suspicious on section. The mass itself was thought to be carcinoma, which was afterward confirmed by the pathologist. Pylorectomy with gastrojejunostomy was done. The patient made an uninterrupted recovery, and when last heard from, six months ago, was doing well.

Let us consider the pros and cons in these 4 cases. In the first there is no definite ground for the supposition of a pre-existing ulcer; as the onset with hæmatemesis one year before is not against a beginning cancer. In the second, the history of "indigestion" for six years *suggests* the possibility of ulcer, but the recentness of the hæmatemesis (fifteen months) and the vomiting (four months) all point to a developing carcinoma. In the third case we have no previous history to aid us, and the most we can say is that there was an old ulcer which might have developed into cancer if the patient had survived, but was not definitely malignant at the time of his death. The fourth case is undoubtedly one of carcinoma, which,

from the preceding history, probably developed upon an old ulcer, though from the size and character of the mass the early symptoms might have been due to a slow growing scirrhus cancer.

Hence in our series there are only 3 out of 82, or, better, 4 out of 83 cases (4.8 per cent.) in which there is any reasonable possibility of a simple ulcer having developed into a malignant one. Fenwick found only 2 out of 89 ulcer cases (2.2 per cent.) which showed evidence of malignancy. Contrary to our belief, there are many who consider that a large proportion of cancers of the stomach develop from a pre-existing benign ulcer. Riegel believes it to be a frequent occurrence, while Zenker maintains that the majority of carcinomata originate from ulcer. According to Lebert 9 out of every 100 cases of carcinoma of the stomach are due to ulcer. Graham,[2] Mayo's associate, found in 135 cases of cancer an ulcer history in 60 per cent. Rosenheim found such a history in only 6 per cent. of the cancer cases, and Barling and Hirschfeld in but 5 per cent.

Sticker pointed out that in those rare cases of carcinoma in which free hydrochloric acid persisted the origin was in a benign ulcer.

Associated Conditions. These are numerous. Tuberculosis occurred in 4 cases; in two the involvement was of the peritoneum and lungs, and in two general. Cirrhosis of the liver was present in 3 cases and obscured the clinical diagnosis, the hæmatemesis being ascribed to a ruptured varix. Jaundice was present in 2 cases from involvement of the gall-ducts in adhesions: in 2 further cases gallstones were found. It is interesting to note that Case 75 was at first thought by the family physician to be one of gallstones on account of the site of the pain; the subsequent diagnosis, however, was cicatricial stenosis of the pylorus. At the exploratory operation the cicatrices were found to be in the duodenum. The patient is now perfectly well. Two cases were complicated by aneurysm of the aorta, 1 of these died from hemorrhage from erosion of a vessel in the gastric ulcer. Two patients had chronic nephritis, and 2 had arthritis deformans. What part these processes played in the etiology is difficult to estimate. There were many other associated conditions (erysipelas, strangulated hernia, pyosalpingitis, etc.). The most important one was typhoid fever, in the course of which sudden pain in the epigastrium and profuse hemorrhage with signs of peritonitis led to an exploration for "perforation," with negative result. One day later, at autopsy, an acute gastric ulcer, with erosion of a vessel, was found.

Duration and Course. The chronicity of the disease is well recognized. The usually accepted time-limit is from three to five years. Sixty-four of our cases had suffered from periods varying from two months to twenty years, the average being 4.1 years. Cases are on record of longer duration. Thus, Habershon reports a case of forty-six years, and Brinton one of thirty-five years' dura-

tion. Lebert found ulcer to be latent in 15 per cent.; of less than one year's duration, 18 per cent.; one to six years, 46.5 per cent.; six to twenty years, 18 per cent.; and twenty to twenty-five years, 2.5 per cent.

TREATMENT. *Medical Treatment.* The treatment followed in the medical wards is similar to that in vogue elsewhere. First of all, absolute bodily rest is insisted upon for a period of from three to six weeks. The stomach is given either complete rest (*i. e.*, rectal feeding for a period varying from five days to two weeks, according to the severity of the case), or partial rest is afforded by the blandest diet—thin soups, whey, milk, etc. Some clinicians, as Fournier, insist upon rectal alimentation for two weeks or even one month. The bowels are regulated, and the anæmia, if present, treated by iron or arsenic. The hyperacidity is counteracted by large doses of carbonate of magnesia, and occasionally by gastric lavage. This latter was instituted in a few cases only, but with marked benefit, even in those without dilatation or obstruction.

Surgical Treatment. The indications for surgical treatment, according to Mayo Robson, are: 1. Perforation. 2. Perigastric adhesions, as pyloric obstruction or hour-glass stomach. 3. Ulcer, intractable to prolonged medical treatment. 4. Recurring and profuse hæmatemesis. 5. Certain cases of acute hæmatemesis.

Let us consider these various indications:

Perforation. All now are agreed upon the advisability of immediate operation for this fatal complication. The great strides that have been made in this branch of surgery are very encouraging. Thus, Weir and Foote, in 1896, gave the mortality in perforation cases treated surgically at 71.5 per cent., while four years later Tinker[20] found that it had dropped to 42.6 per cent.—indeed, in those operated upon in the first twenty-four hours the mortality was only 25 per cent. Mayo Robson[21] in the same year in 429 collected cases found the mortality to be 35 per cent. Brunner,[16] in 1903, gave the mortality as 48 per cent. in gastric and 80 per cent. in duodenal. Of course, statistics collected from literature give an exaggerated idea of the good results of operation, as the majority of the favorable cases are published; nevertheless, the mortality from perforative ulcer is becoming rapidly less—in direct ratio to the number of cases that are early handed over to the surgeon.

Hemorrhage. In this hospital only one case of hemorrhage from gastric ulcer has been treated by the surgeon. This was Case 62 (the typhoid fever patient), who had developed sudden abdominal pain, hemorrhage, and signs of perforation, but in whom the source of the bleeding was, however, not discovered until after death. Dieulafoy[15] advises surgical intervention, if the hemorrhage is as much as half a litre, and *insists* if the bleeding is repeated in twenty-four hours. Moynihan[22] divides the cases into two classes: 1. Acute ulcer, in which operation is *not* indicated except in a few

cases where hemorrhage is both copious and recurrent. 2. Chronic ulcer, where operation is indicated in (*a*) a group of cases where bleeding is copious and transient, and (*b*) frequent recurring hemorrhage, which is now and then profuse.

While concurring for the most part with the previous writers we cannot agree with a recent statement[23] that "it would appear justifiable to perform gastroenterostomy in every case of gastric ulcer in which, in addition to the other clinical data, gastric hemorrhage is observed." Rodman collected, in 1900, 32 cases of acute gastrorrhagia, with 40.6 per cent. mortality, and 31 cases of chronic gastrorrhagia, with 19.3 per cent. mortality. Mayo Robson,[21] in 1900, found a mortality of 64.2 per cent. in operative cases, as compared with 5 to 10 per cent. treated medically. Moynihan,[8] in 1904, quotes 1 death in 15 cases treated for acute or recurring hemorrhage (6.6 per cent.). Robson, we believe, takes the more conservative stand when he states that medical and general treatment is successful in arresting hæmatemesis in 93 per cent. of the cases, hence should be given a fair and complete trial for at least a few hours.

In the small number of cases where surgical intervention is advisable, Finney's gastroduodenostomy or a posterior gastroenterostomy seems the operation of choice.

Perigastric adhesions were present in 11 of the 23 gastric cases at autopsy or operation (48 per cent.), and in 3 of the 4 duodenal cases (75 per cent.). According to Robson, these are present in 5 per cent. of all necropsies and in 40 per cent. of gastric ulcer cases. Hale White[25] gives the figure in the latter case at 45 per cent., while Fenwick found the stomach adherent in 46 per cent. White has outlined the points in the diagnosis of this complication. There is present very severe pain, more or less constant, not increased by food, though often paroxysmal, and often persisting for years. Dilatation of the stomach or symptoms of gastric ulcer may or may not be present. Vomiting or emaciation may be absent, as the digestion is good. Local tenderness or even an indefinite mass from matting together of the viscera sometimes occurs. In such cases operative measures should be undertaken. White succeeded in curing 3 out of 5 cases.

Comparison between Surgical and Medical Results. Hardly a sufficient number of cases treated by surgical interference have been followed for a sufficient period to justify a comparison with the results of medical treatment. Mayo Robson had a mortality in 1901 of 5 per cent; W. J. Mayo, of Rochester, Minnesota, 6.5 per cent.; Peterson and Merchol, 11 per cent. Moynihan, in 1904, among 100 cases treated by gastroenterostomy alone, had a mortality of but 2 per cent. These figures all compare favorably with those resulting from medical treatment, the mortality of which varies between 10 and 20 per cent.

The 82 cases treated in the Johns Hokpins Hospital may be tabulated as follows: died, 24 (29.3 per cent.); well, 14 (17.1 per cent.); improved, 39 (47.6 per cent.); unimproved, 3 (3.6 per cent.); not treated, 2 (2.4 per cent.). The term "well" was applied only to those cases which had been free from all symptoms of ulcer for a sufficient time to warrant the hope that the disease was arrested. The large number of deaths (29.3 per cent.) is difficult to explain until one realizes that 11 of these cases were not treated for the ulcer, but died from some intercurrent affection, the ulcer being recognized only at autopsy. The mortality of 69 treated cases was 13, or 18.8 per cent. Cordier says that ulcer is four or five times more frequent in women than in men, but that the mortality during the latter is much greater. In our series there were 16 deaths among the males to 8 among the females—*i. e.*, the proportion was 2 to 1, which, however, corresponds to the incidence in our series, so that the mortality in both series is practically equal. Of the 58 cases that left the hospital during the past fifteen years we sent out a circular letter and received replies from twenty-six, while eleven letters were returned unclaimed.*

The after-result of these twenty-six for an average period of five years is seen in Table IV.:

TABLE IV.

At discharge.		At an average of 5 years.			
		Recurrence.	Death.	Well.	Total.
Died,	24 = 29.3 per ct.	0	0	0	0
Well,	14 = 17.1 "	4	2	5	11
Improved,	39 = 47.6 "	3	4	8	15
Unimproved,	3 = 3.6 "	0	0	0	0
Not treated,	2 = 2.4 "	0	0	0	0
Total,	82	7	6	13	26

* CIRCULAR LETTER.

DEAR .....

We are interested to know what your condition has been since discharge from the hospital in . Will you kindly answer the following questions:

1. Pain in stomach, if any?
   a. Location.
   b. Character of pain.
   c. Relation to food.
2. Vomiting, if any?
   a. Amount, large or small.
   b. Vomiting of blood, quantity, dark or bright.
3. Passage of blood in stools?
4. Have you been in any hospital since discharge from Johns Hopkins? If so mention hospital and about date of admission.
5. If patient be dead will nearest relative kindly report from what cause and under whose care (physician or hospital)? Were symptoms of stomach trouble still present?

Yours truly,

.....

In other words, of 26 patients since discharge there have been 6 deaths (23 per cent.), 7 recurrences (26.8 per cent.), and 13 permanent cures (50 per cent.) for periods ranging from six months to thirteen years. Of the 6 deaths, the cause was gastric in 2, non-gastric in 2, and unknown in 2. Greenough and Joslin publish 55 per cent. cured, 25 per cent. relieved, 13 per cent. dead, and 6 per cent. otherwise at discharge. Their after-results are seen in Table V.:

TABLE V.—GREENOUGH AND JOSLIN'S 114 CASES.

	At discharge.	Recurrence.	Death.	Cure.	Total.
Cured,	63 = 55 per ct.	28	8	27	63
Relieved,	29 = 25 "	10	4	15	29
Deaths,	15 = 13 "	...	...	...	15
Otherwse,	7 = 6 "	3	3	1	7
Total,	114	41	15	43	114

The latter table shows recurrence in 50 per cent. as compared with 85 per cent. of Welch, 74 per cent. of von Leube, 75 per cent. of Ewald, and 50 per cent. of Debove.

Russel,[25] of Birmingham, followed the after-history of 63 cases in females, and found that after a period of two years there was a direct mortality in 2.1 per cent. Recovery in 42.6 per cent. and recurrence in 44.7 per cent. Schulz[27] followed 88 cases of ulcer treated medically, in all of which hæmatemesis was present, and found that there were 9 per cent. deaths and only 64 per cent. permanent cures.

Of our 58 cases of gastric ulcer that received medical treatment, 11 were discharged well, 38 improved, 3 unimproved, and 5 dead—*i. e.*, a mortality of only 8.6 per cent. Eleven cases have been transferred for operation, which is too small a number for comparison with the above.

MORBID ANATOMY. For observation we had 23 cases at autopsy and 4 at operation. 23 were gastric and 4 duodenal.

CONDITION OF THE ULCER. The ulcer was more or less active in 19 cases of gastric ulcer and 3 of duodenal. Cicatrization had occurred in 3 gastric and 1 duodenal. In 1 case the condition of the gastric ulcer was not stated. That is to say, there were 22 ulcers to 4 scars—a ratio of about 5.5 to 1, rather contrary to Welch, who considers that the scar is at least twice as frequent as open ulcer.

As to the *number*. In 20 cases—16 gastric and 4 duodenal—there was but one ulcer; in 1 case there were two ulcers; in 1 three ulcers, and in 3 (11.1 per cent.) there were several ulcers. Brinton believes that multiple ulcers are found in 20 per cent.

The particulars as to the site, presence of adhesions, local or general peritonitis, etc., are given in detail under the various clinical sections.

CONCLUSIONS. 1. Gastric ulcer is rare in the Johns Hopkins Hospital as compared with cancer, the respective incidences being 1 to 225 and 1 to 56 general admissions.

2. Gastric ulcer in our series was as common in the male as in the female. In the male the percentage of greatest frequency was between the ages of forty and fifty—a decade later than usual.

3. Ulcer was in our cases relatively more frequent in the colored race and among Germans.

4. Vomiting occurred in 85.3 per cent.; pain in 82.9 per cent., and hæmatemesis in 75.6 per cent.

5. Great loss of weight may be present; thus, in 36 cases there was a loss of more than ten pounds, and in 9 of forty pounds or more.

6. Our statistics would indicate that hyperchlorhydria is not so constant as usually maintained; it was present in only 17.6 per cent. of our cases.

7. The blood picture is one of chloranæmia as seen from the average count (hæmoglobin, 58 per cent.; red blood corpuscles, 4,071,000; white blood corpuscles, 7500 per c.mm.).

8. Hemorrhage was the cause of death in 8.5 per cent. of the total number of cases, and in 29.5 per cent. of the fatal cases.

9. Perforation is rare (3 cases, or 3.6 per cent. of our series). General peritonitis occurred in but one instance (1.2 per cent.).

10. Ulcus carcinomatosum is rare—at least 4.8 per cent. of our series.

11. Operation is indicated in all cases with perforation or perigastric adhesions, and in cases of copious or recurring hemorrhage, when medical means have failed after a fair trial.

12. The mortality of the series was 29.3 per cent.; in the cases, however, who received treatment there was a mortality of only 18.8 per cent.; in those receiving medical treatment alone, 8.6 per cent.

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INTERMITTENT HYPERCHLORHYDRIA AS AN OCCASIONAL CAUSE OF RECURRENT VOMITING IN CHILDREN.

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The object of this paper is to direct attention to the curious symptom-complex called cyclic, periodical, or, better, recurrent vomiting of children, with the idea (1) that the condition is not as rare as is generally supposed; (2) that it is relatively easy of diagnosis, an examination of the vomited matter being most important; (3) that at least in some cases the gastric irritability is due to an intermittent hyperchlorhydria, a secretory neurosis, causing the sudden hypersecretion of free HCl and gastric juice.

Vomiting is the main symptom of the malady. The subject, except for the attacks, is in perfect health. The vomiting attacks occur at irregular intervals, begin with slight premonition, continue usually a few days, and cease suddenly, leaving the patient with an unimpaired appetite and digestion.

A similar group of symptoms has long been recognized in adults. In mature life gastric pain is a most prominent feature, while children suffer more from nausea.

The condition in children was first recognized by American authors, although a suggestion as to its existence was first published by Gee. In 1893 I attended a little girl who was harassed by repeated and dangerous attacks of vomiting, lasting several days, and appearing without adequate exciting cause or dietetic error. I reported the case, under the title of "A Gastric Neurosis in Childhood," to the American Pediatric Society. An interesting discussion followed, showing that many of the members had seen these repeated prostrating attacks of vomiting, that they did not consider them ordinary gastric indigestion or gastritis, as they appeared suddenly in children with normal digestion, carefully fed, and the illnesses terminated abruptly, and were separated by intervals of perfect health.

Since then numerous cases of recurrent vomiting have been reported, and the condition is mentioned in most English and Amer-

ican text-books. Either the disease is rare or unrecognized in Germany, as Heubner in his new book entirely ignores it.

An explanation as to its cause has been diligently sought, principally through urinary analyses, and in my opinion no satisfactory theory as to its etiology has been advanced. Probably several phases of disturbed or faulty metabolism may provoke the vomiting.

Marfan gives an accurate description of the condition under the title of "vomiting and acetonuria." He evidently regards it of common occurrence, as he cites twenty-five personal cases. He found the vomiting associated with the odor of acetone in the breath and acetone in the urine, and regards the malady as the result of an intoxication, the acetone being the witness or companion of the toxic product. The expression of the malady is through the stomach, giving rise to persistent and dangerous vomiting.

Edsall believes that recurrent vomiting is sometimes the result of an acidosis, a cryptogenic acid intoxication analogous to the fatal intoxication seen in diabetic coma and other exhausting diseases. His proof is that the odor of acetone is present in the breath and that acetone, diacetic acid, and oxybutyric acid are present in the urine, but only during an attack. The acetone is not the cause of the symptoms, the vomiting being due to the presence of an excessive amount of acid, producing a reduction of the alkalinity of the tissues and body fluids and the loss of alkalies in the excretions, the acids carrying off the alkalies in combination.

Edsall recommends giving large doses of a soluble alkali at the beginning of an attack, to combat the acid intoxication, and certainly his treatment is the most effective known, as the vomiting resisting all other known modes of treatment will occasionally disappear or be prevented, after the administration of large doses of bicarbonate of soda.

Rachford found poisonous leukomaines in the urine, and believes the condition a lithæmic gastric neurosis, allied to migraine. In his cases the vomiting disappeared and was replaced by migraine in adolescence. It may be remembered that migraine is often caused by hyperchlorhydria.

Holt regards recurrent vomiting as a nervous explosion, analogous to migraine in the adult, due to faulty metabolism.

It is to me remarkable, in an illness whose main symptom is vomiting, that so few accurate analyses have been made of the vomited matter. Testing the ejecta would seem to me to be the clue to correct diagnosis and treatment. However, Holt reports the usual findings as of a fluid containing mucus and free HCl. In four of my personal cases the fluid vomited was apparently pure gastric juice, containing an excess of free HCl and mucus, and in the fifth case the hyperacidity was due to combined chlorides.

It is, therefore, probable that *some* cases of recurrent vomiting are the result of an intermittent form of hypersecretion of highly acid gastric juice.

If recurrent vomiting in chlidren is sometimes due to a sudden hypersecretion of irritating gastric juice, the symptoms are far more dangerous and prolonged than in analogous conditions in adults. The free HCl primarily causes spasmodic contractions of the pyloric valve, increased peristalsis of the stomach walls producing gastric distress, nausea, and frequent vomiting. Often blood is mixed with the ejecta, due to traumatism of the stomach walls from violent retching. The vomiting is followed by rapid emaciation, great cardiac weakness, and on rare occasions death.

The disease usually begins in early childhood, from the first to the sixth year. All of my cases were of the better class of society—nervous, high-strung children of average physique. None of them had been debilitated by any exhausting malady. All were fed with an excess of caution. In none was there any flagrant dietetic error. The prodromal symptoms were insignificant—languor and slight fever. Many of the attacks arose out of a clear sky, beginning suddenly, with no adequate cause, ending abruptly, leaving the digestion undamaged, with weeks or months of excellent health.

I will give short histories of five of my personal cases. All were girls. Of 20 recorded cases, 12 were girls, 8 boys.

Case I.—Margeret R. Both parents in good health. A baby sister died at fifteen months, after several days of uncontrollable vomiting.

Child born on October 20, 1899; suckled eleven months. After weaning there followed a period of anorexia, lasting four months. During this time the baby would not take food from a bottle, cup, or spoon, nor suckle a wet-nurse. For four months the father, a physician, laid the baby on its back, held its hands, and forced down food into its mouth with a large pipette, the child meanwhile expressing its aversion to its meals in the most violent manner. It digested what it swallowed, and slowly improved. At three years it was brought to the city in superb health after a summer in the country. In a week she had an attack of vomiting lasting forty-eight hours. During that time there was a temperature of 101°, a slightly coated tongue, and very abrupt cessation of all symptoms, save there was mucus in the stools for a few days.

After this the illnesses recurred every six to eight weeks. The little girl would go to bed perfectly well, would commence vomiting at night, and would vomit every half-hour to two hours until the attack terminated, usually two to three days, absolutely nothing being retained in the stomach. During the illnesses there was thirst. The temperature was 100° to 101°, the tongue gradually becoming more coated. The skin and breath had a curious fruity odor—acetone. The abdomen was never tender, but always

retracted, the contour being due to empty intestines and general asthenia. The bowels were constipated; an enema would bring away a well-digested stool. The vomited matter was, first, food debris, later one to two ounces of pure gastric juice. In an attack periodically the child would become restless from gastric distress, would vomit, and be easy for a while. Suddenly the child would look better, and take and retain its ordinary meals. In spite of a restricted diet and semistarvation, the attacks continued and grew worse. One in April lasted four days. There was noticeable emaciation and exhaustion; the pulse was weak and irregular; sighing respiration and cold extremities. Urine examined; no albumin, casts, or sugar.

In May, 1903, occurred the worst and final attack. I saw the child after it had vomited two days. The surface was cold. Temperature 102°; pulse 160, very feeble; patient delirious; there was a noticeable odor of acetone in the breath. The vomiting was projectile; the ejecta contained mucus and streaks of blood. The girl was in a dangerous condition of cardiac weakness from inanition and protracted vomiting. An examination of the ejecta showed it to be gastric juice, acidity 18, due to free HCl; mucus, blood, and shreds of the stomach walls were also present. A hypodermic of strychnine was given, and first 10 grains and later 20 grains of bicarbonate of soda in water every hour by the mouth. After the alkaline treatment, vomiting occurred only once (200 grains of bicarbonate of soda were given in twelve hours). A remarkable amelioration was soon seen in the child's general condition. The gastric distress ceased, the odor of acetone disappeared, the skin became soft and warm, and the next day she was put back on her usual diet. The urine continued acid until the second day, although 400 grains of soda were given in thirty-six hours. For eleven months this child was given soda in sufficient doses to keep the urine slightly acid. One day in the winter she vomited for a day. The doctor gave 20 grains of soda every hour, and fed her beef-juice and malted milk. The food was retained; the vomited fluid was hyperacid gastric juice. No real recurrence has taken place in eleven months. To my regret no examination of the urine for acetone or diacetic acid was made, but the odor of acetone was evident in the breath.

This is a case of Edsall's acid intoxication causing a hypersecretion of gastric juice. The alkaline treatment was brilliantly successful in arresting the vomiting and preventing a recurrence for nearly a year.

Case II.—Helen S., daughter of a physician. Pertussis at three years; at five years developed a craving for bicarbonate of soda, asking for it and preferring it to sweets. During the summer of 1903 she was in good health, eating freely of fruits.

In September, 1903, while taking a drive, she began to cry, and vomited half-digested food. Temperature at first 99.6°, later

normal. The vomiting continued four days. During this time the child was much distressed from nausea, and threw up every hour one to two ounces of bitter, greenish or yellowish fluid containing mucus, and on two occasions blood. The tongue was coated, and the breath had the odor of a sour stomach; abdomen retracted; bowels moving only after enemata. In the last two days the patient sunk into a condition of grave prostration, with a rapid, weak pulse. Ten pounds of flesh were lost in this illness. After the retching ceased the child had a ravenous appetite, and digested well a very restricted diet.

October 25th. For no apparent reason the vomiting recurred and lasted seventy-two hours. The father noticed much more liquid was vomited than was swallowed.

January 5th. Another attack lasting four days. The illness was characterized by paroxysms of intense abdominal pain, relieved by vomiting. On the fourth day the child was carefully examined. There was no odor of acetone in the breath. The ejecta contained no free HCl; total acidity 40, due to combined chlorides. In exceptional cases with symptoms of hyperchlorhydria the gastric contents show a high acidity due to combined chlorides, not to HCl.[1] The father gave a heaped teaspoonful—90 grains—of sodium bicarbonate at a dose in water every one and one-half hours; vomiting occurred only once after the alkali. (In this case the soda was given in too large a dose and might have irritated the stomach.)

February 10th. Urine analyzed in a period of apparent health; acetone present; no diacetic or oxybutyric acid, showing very clearly that the acetone in this case was not due to inanition from an exhausting malady, but was a premonition of an impending systemic intoxication. I told the father the girl would soon be sick again, and going back to lunch he found her crying from pain in her stomach. He gave her a large dose of soda, and the distress disappeared. From February 10th to March 18th he kept the urine alkaline. On March 18th the father was ill with la grippe, and soon after the daughter sickened, with hoarseness, nasal discharge, and fever. On the third day the influenza was complicated by frequent vomiting, lasting four days. The acidity of the stomach contents was 40, due to combined chlorides; no free HCl. The vomiting was not relieved by soda, which was again given in too large a dose. On her recovery the child was taken to an oculist, who found some error of refraction and put on glasses. There was so much anæmia and debility from this illness that the child was taken South.

In this case the gastric irritability was due to combined chlorides.

Case III.—Helen W., aged nine years, always constipated in sickness and health; otherwise, a well-developed, handsome girl. In infancy was fed on peptonized milk and suffered from consti-

[1] Stockton, p. 315.

pation, colic, and a slow gain in weight. First attack of vomiting, lasting seven days, occurred at two years of age from improper feeding. From that time, with the exception of her sixth year, she has had vomiting, attacks lasting from two to fourteen days at intervals of six to twelve weeks. Diet made no difference in preventing attacks; between them her digestion was perfect. Her illnesses were more frequent in winter than in summer. They were occasionally precipitated by fatigue after a long walk. They would sometimes cause alarming prostration. The family physician said the vomiting was always preceded by aggravated constipation.

July, 1903, was spent at the seashore; the patient grew strong and vigorous. Immediately on her return she was sick, and vomited thirty times in eighteen hours. The attending physician gave 10 grains of soda every hour, but the remedy apparently irritated the stomach and increased the retching. Recovery was slow, and the child would not eat for a number of days. After this 10 grains of soda were given three times a day. She was vaccinated in November and vomited for a day.

December 19th. The child began vomiting at 7 A.M.; given 10 grains of soda every hour. Emesis at 7, 8.15, 9.15, 9.45, 10.35, 11.15, 11.56 A.M.; 12.35, 1.05, 1.35, 2.15, 2.55, 3.20, 4, 5.40, 6.30, 7, 7.45, 8.55, 10.35 P.M.

20th. 12.15, 1.15, 5.30, 8.20, 10.45 A.M.; 1.35, 2.30, 3.55, 5.45, 7.05, 7.20, 8.20, 9.20, 9.40, 10.45 P.M.

21st. 12.30, 1.30, 2.30, 2.46, 4.05, 5.05, 5.25, 6.30, 6.50, 7.40, 8.50, 9.40, 10.40, 11.40, 12 A.M.

22d. 1, 1.30, 2, 2.30; attack ceased suddenly at 2.30 A.M. Vomited forty-one times in sixty-seven and one-half hours.

I saw the patient on the third day of the illness at 4 P.M.; rectal temperature 100°; skin cold and clammy; dark circles under the eyes; tongue slightly coated; abdomen retracted, not tender; no odor of acetone to the breath; heart action fairly strong (135); urine scanty. The child was thirsty; always threw up the water swallowed. In fact, it made no difference in the vomiting whether anything were taken or not there was much nausea. The ejecta consisted of an acid, milky colored fluid containing free HCl. Urine contained no albumin, casts, or sugar; not examined for acetone.

The last attack occurred in February. The patient vomited continually for ten days, then a lull of a few hours, in which food was given; then it began again and lasted eighteen days, until her life was despaired of. Her convalescence was so slow that she remained in bed a month. This is the longest and most severe attack I have knowledge of that did not result in death. The alkali was persistently given in suitable doses, but gave no relief. We were never even successful in neutralizing the acidity of the ejecta. The only ray of comfort was given by hypodermics of morphine, which

strengthened the heart and gave some slight respite from the frequent retching.

CASE IV.—Ruth N., an active, very intelligent, and emotional little girl. During infancy she was nursed, but from the age of one month she would often become feverish and vomit continually from twenty-four to seventy-two hours. These attacks were never preceded by constipation or prevented by the most rigid diet. After infancy they were often associated with sore throat. At the age of four and one-half years nasal adenoids were removed. During the summer of 1902 there were two attacks, when every hour or two she would throw up a quantity of clear, sour-smelling, watery fluid mixed with mucus. The child lay motionless in bed, refusing to swallow or to stir. She would literally get well in a minute, go out, eat, and enjoy herself.

Description of Two Attacks.

September 28th. She sickened in the night, with a temperature of 104°, pulse 150, vomiting, headache, and sore throat; the pharynx was not inflamed; bowels moved in the early morning. After forty-eight hours the little girl became much prostrated by her symptoms. The mother took the child out-of-doors at midnight; the nausea disappeared, and she called for food, slept, and was well again.

December 27th. Child languid, complained of headache; temperature 104°; from 6 A.M. to 10 A.M. vomited three times; 10 A.M., pulse 165, temperature 101° at 4 P.M.; the little girl was smiling; vomit matter examined; hyperacid gastric juice; total acidity 10, due to free HCl; 0.365 per cent. of free HCl.

The vomiting did not return, but the patient suffered for a week with fever and left-sided torticollis.

In March, 1903, child had an attack of whooping-cough, in which malady, strange too, although vomiting is expected, it did not occur. This would indicate that these gastric crises were not due to any abnormal stomach reflex, but to some central nervous irritation or intoxication stimulating the centres governing the secretion of HCl. This case is rather atypical, as the gastric crises were associated with high fever. There was also a strong psychical element, as the vomiting would occasionally stop suddenly, by a pleasurable sensation, by the appearance of the father with a new toy, or if the child were taken into the open air.

CASE V.—Katharine C., aged eight years; a very neurotic family history; grandfather and father alcoholic; mother suffered from eclampsia in infancy; an elder sister died of epilepsy; two brothers had infantile convulsions. The little patient herself was rachitic.

First manifestation began at nineteen months, with a convulsion, followed by slight fever and the frequent vomiting of a thin, acid

fluid for five days; sudden cessation of vomiting and restoration of digestion.

Eleven months later a second attack, ushered in by a convulsion, with fever and vomiting for five days, causing grave exhaustion. After this the eclampsia ceased, but the vomiting attacks continued at intervals for two years. Once she brought up a large quantity of fresh blood. She would barely recover from an illness before another commenced.

In January, 1893, I first saw the case. She had had the usual symptoms for ten days, and her parents were alarmed at her dangerous weakness. Temperature 101.5°, pulse 120. Pulse and respiration regular; tongue slightly coated; abdomen not tender, markedly retracted; rectal examination negative; one stool in ten days.

The gastric irritation seemed to recur periodically; there would be a burning in the stomach; nausea. The child would swallow water, and immediately vomit a very acrid fluid.

She was a slender, undersized girl, not emotional, save for these gastric crises; appetite, digestion, and stools were perfect. All the medicine and food that I ordered were vomited. On the fourteenth day the symptoms disappeared, and she ate and slept and played as usual.

In seven weeks another attack occurred, precipitated by a puzzling lesson in subtraction. The vomited matter was pure gastric juice, containing an excess of free HCl. Alkalies did not relieve the symptoms nor neutralize the acidity. The vomiting was evidently due to a recurring hypersecretion of gastric juice, irritating the stomach and pylorus. She was given large doses of chloral and bromide by rectum, and slept with one-half hour's intermission for twenty-nine hours, and awoke with good appetite and digestion. This was the thirtieth attack.

She was taken out of school, given tonics and gymnastics, and remained well for eight months, when I lost track of her.

The dominant symptom of the malady is vomiting coming on spontaneously, with no exciting cause. Some of my cases were feverish, one having a temperature of 104°. Holt says the lack of fever is a point of much diagnostic value—a statement at variance with my experience.

The vomiting crises are not due to acute indigestion, gastritis, or bad feeding. The integrity of the gastrointestinal functions is generally perfect, save for the recurring illnesses. No known plan of dietetics will prevent the attacks. Stomach digestion and intestinal peristalsis are temporarily arrested, and are rapidly restored when the gastric irritability is over. Fatigue, excitement, anxiety, school work are more likely to cause an attack than indulgence at the table. Sea-air, country life, no study, are more likely to lessen the severity and frequency of the attacks than semistarvation and

medication. Nevertheless, the condition is apt to disappear in adolescence.

I would state my opinion that the cause of recurrent vomiting is still unknown, but it is probably a transitory autointoxication, playing with full force on the nervous centres governing gastric secretion and motion, that in some cases the lesion of the stomach is an intermittent form of hyperchlorhydria.

DIAGNOSIS. In any suspicious case the urine should always be examined for albumin and casts, and the odor of the breath and the reaction of the stomach contents noted. The first attack will be taken for indigestion, but the course of the malady and recurrence will clear up the diagnosis. Some cases have been mistaken for meningitis on account of headache, vomiting, constipation, and boat-shaped abdomen, but a patient with meningitis does not vomit pure gastric juice or recover in four or five days.

The patient might give an impression of appendicitis or intussusception, but there is no abdominal tenderness, or tumor, or prolapsed bowel, or blood in the stools. A severe attack will cause the physician the deepest apprehension, as day after day he views the rapid emaciation, great cardiac weakness due to loss of fluids, lack of nourishment, frequent retching and vomiting.

Nearly all cases get well, yet as our knowledge of the course of the disease becomes complete, and its relative frequency becomes evident, an increasing number of fatalities are reported. The post-mortem findings are necrotic changes in the gastric and intestinal mucosa and inflammatory or irritative lesions of the kidney. Yet in the most desperate emergency, when the child is apparently moribund, a sudden recovery is probable. Further, any attack may be the last. Some unknown influence may so invigorate the child's vitality that the gastric crises do not recur.

THERAPY. It is difficult to estimate the effect of treatment in an affection that may terminate spontaneously at any time, and in which long periods of health occur. The alkaline treatment was strikingly effective in at least one of my cases, and should always be tried. In dangerous conditions nutrient enemata, chloral by rectum, and hypodermics of morphine and strychnine may tide you over a dangerous crisis.

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MEDICAL TREATMENT OF GASTRIC ULCER.

By Samuel W. Lambert, M.D.,
of New York.

Ulcer of the stomach has always existed and physicians have always treated it, but an accurate knowledge of its pathology and a rational understanding of its treatment date back less than a hundred years. There is probably a medical treatment of gastric ulcer as old as Hippocrates, and it is customary to credit the Father of Medicine with a knowledge of this disease. It is true that he described a *morbus niger* characterized by black vomit, melæna, darkened skin, pain after eating, and loss of flesh and strength, and that he prescribed a diet of milk, sweet and fatty foods, abstention from wine, salt, and spices, further advising bloodletting and mild cathartics, with avoidance of exercise and exposure to the heat of the sun. But such a description and such alleviative measures apply equally well to certain cases of cancer, or of hepatic cirrhosis, or to other causes of gastric hemorrhage, and it seems doubtful if ulcer of the stomach was known as a distinct disease before the last half of the eighteenth century. At all events, it was the epoch-making publications of Cruveilhier in the 30's which placed this disease on the firm modern basis of pathology, and first gave an opportunity for a scientific therapeusis, which Cruveilhier was also the first to formulate.

The indications for such a treatment depend on the general and local conditions present in those afflicted with the disease. The patient is usually anæmic and, because of partial starvation, below the normal standard of nutrition. In certain cases he is a sufferer from severe nervous symptoms. The stomach, in addition to the ulcer, is the seat of a grave functional disturbance, as is evidenced by hyperacidity, with more or less stagnation of food, and by irritability, with vomiting, nausea, and pain. A serious or even fatal complication in the form of a hemorrhage or a perforation of the ulcer is a possibility and constant menace to the life of the patient. But the principal fact to be remembered in treating ulcer of the stomach is that there is no specific and that the ulcer heals through the power of repair in the body. The therapeutic indications may be formulated thus:

I. To assist nature in the process of repair.

a. By regulating the diet.

1. To protect the ulcer from mechanical injury and consequent further extension of the ulceration.
2. To keep the ulcer at rest.

b. By administration of drugs.
1. To stimulate cicatrization.
2. To cover and protect the ulcer from chemical irritation.
3. To neutralize the gastric acidity whether due to the normal acid or to any of the abnormal acids of fermentation.

c. By improving the general health by careful feeding and hygiene.

II. To prevent loss of flesh and strength by feeding through other channels than the stomach.

III. To combat individual symptoms and complications as they arise.

The two cardinal points in every ulcer cure and regimen are rest and milk diet. The oldest treatment consisted of the regulation of the diet, and dates back to Cruveilhier, who advocated, in 1838, a diet of milk exclusively, to be given in small amounts and at frequent intervals, and milk still remains the best food in these cases. But rest is, perhaps, the more important of the two, and was first proposed in the 50's by Brinton, of London, who made it a feature of the treatment which was formulated by him. But the final adoption of rest as an essential in all ulcer cures is due to the careful presentation of this subject by Leube in the 70's and to the influence of von Ziemssen, which was exerted in a publication made about the same time. All such patients should be put to bed and kept there for such a period that the characteristic pain and tenderness consequent upon the taking of a mild but varied diet shall no longer appear. This means that a sufficient period must elapse to allow the patient to pass through the various stages of stomach starvation, milk diet, fluid diet of broths and milk, diet of fluids, eggs, bread, butter, cereals, and lighter meats. The rest cure for cases of gastric ulcer does not mean such an absolute isolation as is indicated for the more serious forms of neurasthenia. It is sufficient that these patients stay in bed and give up all business cares and worries. They may see their friends and the members of their family in frequent short visits; they may be entertained by reading and games, and they may receive letters and even write short replies. During the "rest" they should receive daily alcohol spongings and baths, and mild forms of massage to the arms, legs, and back will improve their general condition. The duration of this rest cure will rarely be prolonged beyond six weeks.

In addition to this general rest a local rest for the stomach and the ulcer itself is indicated, and this brings one to the second cardinal point: the regulation of the diet. As soon as the "cure" is begun and the patient is settled in bed, a period of absolute abstention from food by the stomach should be inaugurated. Nothing but

water and pieces of ice should be swallowed. The feeling of hunger caused by the stomach starvation becomes blunted within a couple of days, but thirst is very troublesome and water must be allowed. Both the loss of nourishment and the loss of fluid may be supplied in some measure by nutrient and saline enemata, although these are poor substitutes and cannot be continued indefinitely. The first week of such treatment is a severe tax on the patience and the confidence of the patient, but some period of starvation by the stomach seems wise, and even in the milder cases the cure will be more certain because of it.

Nutrient enemata were used in the treatment of gastric ulcer by Brinton in conjunction with his rest cure. They are undoubtedly a great assistance in keeping up the strength of the patient. The method of using is to give the patient daily, in the morning, a high lavage of the colon, say of four quarts of normal saline solution. After a rest of at least an hour the regular nutrient enemata may be begun, and may be continued at intervals of four hours during the day. Three, or at most four, such enemata may be given during the twenty-four hours. Each enema should not exceed four ounces in bulk, and may consist of egg, milk, or meat-broths, or of combinations of these foods. All the foods so used should be predigested to a degree of complete peptonization by means of pancreatic extracts and bicarbonate of soda. There is a widespread habit in practice to use this peptonizing process only for milk, and to add to it only the preparations of meat-peptones which are on the market. The freshly prepared peptonized broths and eggs are as easily made as is peptonized milk, and leave no uncertainty as to the amount given. One avoids also the use of alcohol, which is a prominent ingredient of most proprietary peptone foods. Alcohol is not a necessary part of nutrient enemata, and should not be added except for some special therapeutic reason. If the enemata are not well borne, or if the rectum becomes irritable from frequent use, some preparation of opium may be added, and with this assistance the rectal feeding may be continued longer than might be possible without it.

It will be found if the water returned from the morning lavage be measured that a considerable quantity has been absorbed; this will vary somewhat, but it often amounts to half a pint. Such an absorption tends to diminish thirst and to lessen the amount of water demanded by the stomach. It is wise to make further use of this manner of supplying water to the system, and to inject into the bowel a half-pint of saline each evening an hour or so after the last nutrient enema has been given, with the idea that the whole amount will be absorbed. During the night the rectal feeding should be omitted, the patient be allowed to sleep, and the day's routine as outlined be resumed in the morning.

Gastric feeding may be resumed in mild cases within four days,

but in the severer forms of the disease it will be necessary to wait a full week or more. One should begin before the rectum grows intolerant, and should increase the use of the stomach gradually, while the use of enemata is discontinued in like manner. The first food to be given should be peptonized milk in small doses, at rather long intervals. The amount may be increased and the interval shortened until, by the end of a week, the patient will be getting a quart of peptonized milk during the twenty-four hours, and the rectal feeding may be stopped. The progress of the return to a normal diet should be a steady advance from this point on, with some daily change in order that the patient may feel that each day brings an improvement. About two weeks will be consumed in reaching this stage of the cure. The next four weeks will be spent in taking the patient through all the stages of diet which are characteristic of the feeding of infants during the first two years of life, and much benefit can be gained for patients with gastric ulcer from a study of the principles of infant feeding. The main point to be observed in increasing the diet is to make a single change at a time, no matter how simple, in order that if any distress or set-back follows, the new article of diet or the new method of administration may be ascribed as its probable cause. In this manner it is possible to correct quickly any indiscretion in diet and to prevent serious relapses in the course of treatment. But the success of the whole cure depends more on the first step, the inauguration of a milk diet, than on any other detail of the treatment. The use of milk as an exclusive food is subject to much prejudice from the laity and to some misconception even from the profession. I believe it is possible to secure a milk which can be taken by anyone, and would insist on two main propositions: First, there is no method of furnishing a quickly prepared, palatable peptonized milk, but the peptonized milk which is required for use in a cure for gastric ulcer, whether it will be used in the colon or in the stomach, requires the constant application of moderate heat for two hours for its preparation, and the product has a disagreeably bitter, unappetizing taste. Second, the modification of milk, by the dilution of top milk or cream, can furnish a milk of any desired composition. Milk can be so modified that low percentages of casein can be combined with normal or high percentages of sugar and butter fats. And such modified milks, either raw or peptonized, can be made to agree with any stomach, however peculiar the idiosyncrasy of the patient may be.

The popular modification of milk by mixing it with lime-water or Vichy water gives a clue to a method which has been most successfully used with children. It is not so much the addition of the alkali, though that is a help to modify the curd formation, as it is the mere dilution, which is the essential part. Cows' milk has an average constitution of fat 4 per cent., sugar 4 per cent.,

casein 4 per cent., and its casein has the peculiarity of curdling in large lumps. This last peculiarity is usually considered the cause of the failure of the milk to agree, but the abundance of the curd is an equally important factor in causing milk indigestion and gastric irritation. Simple dilution of the milk removes the cause of the trouble, and the use of an alkali as a diluent tends to modify the size of the curd, but there is a loss by the same process in nutritive value by a like dilution of both the butter-fats and the milk-sugar. It will be found that stomachs which are the seat of ulcer will often bear fats well, and although cane-sugar is at times a source of acid fermentation, it is found that milk-sugar is usually well borne. The problem to secure a milk which will not irritate is, therefore, the same as that which has been solved for the artificial feeding of infants, namely, to diminish the proteid and still keep the sugar and fat percentages the same as in normal cows' milk. So much has been written on this subject of late years that it would be superfluous to go farther into detail. It is useless to leave the modification of the milk to the undirected efforts of the average attendant, trained or untrained, but a definite milk formula should be selected and modified as often as necessary. Another source of error can be found in the widespread fallacy that there is a rapid method of peptonizing milk, the so-called "cold process" of proprietary advertisements. It seems absurd that it is necessary to deny the possibility of peptonizing milk by such a method.

After the patient has reached the point where he is on an exclusive stomach diet of peptonized milk, the first advance is to increase the milk till he is taking two quarts or more a day. The next step will be to reduce the degree of peptonization. During the third week the peptonizing process should be cut down to half an hour or twenty minutes' duration, and by the end of the fourth week the milk can be given raw, though modified in strength if originally necessary. During the third week, also, cooked cereal gruels may be added to the milk as part of the diluent. Barley, wheat, and oat flour may be used to advantage in this way. The fourth week will see added to the dietary crust-free bread, butter and boiled egg. One may add during the fifth week potatoes (baked or boiled), rice, boiled macaroni, custards, chicken, and sweetbreads. The final or sixth week of the cure the diet should be increased to include beef, mutton, green vegetables, light salad without vinegar, cooked fruits, and simple puddings. The patient will be allowed out of bed in the fifth week and even earlier in the mild cases. He should be encouraged to exert himself more and more each day, with the hope that he may resume his regular mode of life, except for certain precautions which are essential to avoid a relapse.

The disease tends to recur in new situations as well as relapse in the old, and the rules for a prophylactic diet and mode of life for these cured patients are most important, and their observance

must be insisted on for a long time. Such a patient must avoid frequent eating and drinking, and he must not overdistend the stomach with an excessive amount of food at any meal. He must eat slowly and masticate the food thoroughly. All hard food, like bread-crusts and fruit-seeds, and tough vegetables or meat-fibres, must be omitted in order that there may be no mechanical injury to the gastric mucous membrane. Alcohol in all its forms, spiced and highly cooked sauces and condiments, and even an excess of common salt are to be avoided in order that there may be no excessive chemical irritation of the stomach. Even after the patient has returned to work he must avoid overfatigue, both physical and nervous, and he should abstain from long journeys where he cannot control his diet and mode of daily life.

A specific action in the cure of gastric ulcer has been attributed to certain alkalies and astringents and to two drugs of widely different qualities, namely, the subnitrate of bismuth and olive oil. The alkalies would seem to have no more than antacid and possible laxative action, unless, perhaps, the insoluble calcium carbonate which has been suggested as a substitute for bismuth. The principal astringents which have been used are the perchloride of iron and the nitrate of silver. Both these drugs have a similar degree of astringent action by which the granulating surface will be stimulated to cicatrize, but the silver seems to have given the best results. The nitrate of silver solution must be introduced with a tube in order to reach the stomach in an active condition before it is precipitated as an inert chloride. This alleged disadvantage of the silver salt would seem to be a distinct advantage, not that the nitrate becomes inert, but that by doing so it renders inert a certain amount of the hydrochloric acid of the stomach juices.

The use of bismuth in the treatment of gastric ulcer was suggested in the 60's by Budd and Trousseau, but the definitive claim for a specific bismuth cure has been put forward particularly by Fleiner. Fleiner's mode of procedure is as follows: To wash out the fasting stomach each morning until the water returns clear and is free from an acid reaction, and then to introduce through the tube six ounces of water, with which 2½ to 5 drachms of subnitrate of bismuth have been thoroughly mixed. As soon as the tube is withdrawn the patient should lie down and rest quietly in such a position that the bismuth will gravitate toward the supposed site of the ulcer. After half an hour's rest the patient should be given his breakfast. The result of such a treatment given daily should be immediately apparent in a diminution of the stomach distress and cessation of the pain, nausea, and vomiting. The time to begin the use of the bismuth is coincident with the return of gastric feeding after the initial period of starvation is ended, and should be continued for at least two weeks. Fleiner does not claim that the rest and the regulation of the diet can be omitted;

on the contrary, he states particularly that the bismuth is an aid to the regularly accepted routine as already detailed. Fleiner does claim for the bismuth cure, however, both an antiseptic and astringent action, as well as a direct action to reduce the hyperacidity in addition to the mechanical protective effect of the large doses. Ewald, in the first edition of his *Klinik*, suggested the substitution of the insoluble carbonate of lime for bismuth, and Pariser has recently reported the use of a chalk, talcum, and magnesium mixture in place of bismuth, and with excellent results. The opponents of the bismuth cure have insisted on the disadvantages of its constipating effects, of its discoloring the stools, causing a possible oversight of slight melæna attacks due to small hemorrhages, and of the dangers of using the stomach tube in all ulcer cases. So far as the use of the tube is concerned, it is not an essential, for the bismuth mixtures can be given by the mouth direct, and it has been so given by Fleiner and others, but the other criticisms are not without their significance and weight.

The olive oil cure of Cohnheim is applied in the same manner as is the bismuth cure. The oil is introduced through the tube into the fasting stomach after a moderate lavage, before breakfast, in quantities of from two to four ounces, or it is given before the three principal meals without the use of the tube in doses of from one to two ounces. Cohnheim claims that the oil forms a protective coating to the ulcer, relieves the pain and vomiting and the tendency to pyloric spasm, allows the patient to be fed, and itself acts as a food, and finally that it checks the secretion of acid. His reports would prove that the milder cases can be treated under this oil regimen by simply adding a regulated diet and without any attempt at a rest cure.

Both of these drugs may be given direct by mouth, and have been so used by their adherents, but they require the use of the stomach tube to apply them to the best advantage. This will raise an immediate opposition on the part of many clinicians who have a decided reluctance to introduce a tube into a stomach known to be the seat of an ulcer. It is a difficult task to decide what degree of danger attends such a procedure. If ordinary precautions are taken not to distend the stomach, and not to get too great a pressure by limiting the height of the funnel to the level of the head of the patient, one can ignore the possibility of a perforation of an ulcer. The likelihood of causing a hemorrhage is quite another question. It is doubtful if every clinician has not at some time washed bright blood out of a stomach as a result of the straining and retching due to the irritation of his siphon tube. Such accidents are regularly slight and of no consequence, but at least two authorities have recorded fatal hemorrhage which they believed to be due solely to a gastric lavage. It would seem wise to try the gastric lavage in any case where it was desired to give the patient the

benefit of a thorough bismuth or oil cure, and only to desist upon the occurrence of bleeding as a consequence of the treatment. Of course, cases which have been bleeding spontaneously in the recent past would also be excluded from such procedure.

The treatment of special symptoms and complications forms an interesting and important chapter in the history of this disease. The local subjective symptoms of nausea, vomiting, epigastric distress and pain, are best treated by the treatment of the ulcer itself. They usually disappear during the starvation period which should inaugurate every cure. When they return during the course of the treatment they are to be considered as danger signals that the treatment has progressed too rapidly and that that individual case must be set back to an earlier period of the cure. Nevertheless, these symptoms may require other remedies for their relief than the bismuth, silver, oil, etc., which have been discussed. Antiemetics will often prove of benefit, and anodynes, as codeine, cocaine, and anæsthesin will often control attacks of gastralgia, while morphine should be avoided because it tends to increase acidity. The regular habit of these patients, especially those who have a condition of hyperacidity, is constipation. The most efficient drugs to combat this condition are the alkaline salines, prominent among which Carlsbad water and salts must be mentioned. Such an aperient given each morning before breakfast is usually sufficient. If there be any serious accumulation in the colon, it may be relieved by the use of oil or water enemata. The conditions of thirst and of hunger which are largely due to the treatment itself have been discussed before. The condition of anæmia will require general treatment, and iron will be needed. One can use the pyrophosphate with considerable confidence of having no irritating effect. It may be used during the rest-cure treatment as soon as the patient has begun to add other articles of food to his milk diet.

The most constant accompanying gastric complication is an hyperacidity. This may be overcome in a purely symptomatic way by neutralizing the acid, or the more permanent method of modifying its formation may be adopted. The secretion of the acid may be checked and in large measure controlled by the use of atropin or belladonna. The same result may be secured by the use of bismuth, of oil, and of fatty foods generally, or by irrigations of silver nitrate, as has been already described. The adherents of the oil cure of Cohnheim claim that this simple treatment will overcome most cases of pyloric spasm, even when associated with tetany. Such a procedure demands a trial for no other reason than for its simplicity alone. Two other complications are purely surgical in their treatment: the late complication of gastric ulcer, a cicatricial pyloric stenosis, with its consequent motor insufficiency, and a perforation of the stomach, with peritonitis. Surgery can cure the first, and it is a modern triumph that some degree of suc-

cess is offered in the second, in which form of perforative peritonitis von Ziemssen thirty years ago could only advise as complete an euthanasia as possible.

There remains to be considered the treatment of hemorrhage from the site of an ulcer. This complication may present itself in different degrees of severity. The mildest cases of bleeding in which the amount of blood lost is insignificant may be considered as mere incidents in the course of a disease which is running along smoothly. After any loss of blood which amounts to more than the merest shreds or stains in mucus, discoverable perhaps only by careful chemical analysis of the stools, it is wise to take precautions to avoid irritating food for a day or so. If the blood can be measured by the ounce, a period of fasting should be insisted on as an initial therapeutic measure. This complication is often the first symptom to make these patients realize the severity of their disease, and the therapeutic measures of rest, starvation by stomach, and rectal feeding are frequently begun shortly after a more or less severe attack of hæmatemesis. If the hemorrhage is not repeated, even if it amounted to a pint or more, the regular routine treatment may proceed without a setback. The hemorrhage may become continuous, or it may be repeated at short intervals in spite of treatment, and may demand surgical interference within a few hours. The bleeding may occur at longer intervals, and possibly be associated with each attempt to begin gastric feeding. Such cases present the most difficult question for decision, whether to call on the surgeon for help or to continue medical treatment. A single large hemorrhage, causing death in a few minutes, is also a possibility, and such a complication is, of course, beyond either medical or surgical help.

The medical treatment of active gastric hemorrhage is simple, but often inefficient, and is directed to giving the blood an opportunity to coagulate, and thus to plug the bleeding point. Absolute rest, local application of ice to epigastrium, and the giving of pieces of ice to be swallowed whole, are of some benefit. The stomach should be allowed to contract, and if it be filled with clots a stomach tube should be inserted and the organ emptied. There is only one drug which is of service: the hypodermic use of morphine controls the restlessness and air hunger, keeps the patient quiet, and supports the heart action. The use of alcoholic stimulants is contraindicated, and cardiac whips are decidedly injurious. The loss of fluid and the consequent thirst may be overcome by means of saline enemata. A pint of normal saline solution may be injected into the rectum at hourly intervals, with a fair prospect of its complete absorption, and such enemata may be repeated with advantage for ten or more consecutive hours. A due appreciation of the probabilities of the success of surgery in these cases of hemorrhage, and the utter helplessness of medicine, and of surgery also, unless

the case is undertaken early, will change the medical habits and improve the surgical results of the past.

During the past ten years there have been 52 cases of gastric ulcer admitted to the wards of the New York Hospital. Of these 35 were treated in the medical and 17 in the surgical wards. Of the 52 cases, four were relapses in cases previously treated, so that there were fifty-two admissions of forty-eight persons. Of the 52 cases, eleven died, which is a mortality of 21.15 per cent. in all cases.

Of the 35 cases in the medical wards 30 had hæmatemesis, two of whom died; three abandoned treatment in from two to four days, and were discharged unimproved, and the remaining two were cases of relapse occurring without hemorrhage. The details of the 4 cases of relapse are as follows:

Case I. was operated on for general peritonitis caused by a perforated gastric ulcer in November, 1895, and was treated in the medical division for a relapse without hæmatemesis in October, 1896.

Case II. was cured by operation for a general peritonitis, due to a perforated gastric ulcer, in February, 1898, and was treated in the medical wards for a recurrence without hemorrhage in October, 1898.

Case III. was discharged improved from the medical wards after thirty-nine days' treatment in February, 1902, and died in the surgical service after an operation of gastroenterostomy to relieve a cicatricial pyloric stenosis, in September, 1902.

Case IV. was discharged improved after thirty days' treatment in the medical division in October, 1897, and returned for treatment in March, 1899, but left dissatisfied four days after admission.

Of the 17 cases admitted, or transferred to the surgical division, 3 were admitted moribund, suffering from a general perforative peritonitis, and all died without interference. The 14 cases operated on should be classified as follows: 1 case died after an operation to relieve a cicatricial stenosis, as stated above; 2 cases were operated on for "gastralgia," and in both healed ulcers only were found; both were discharged cured; 3 cases were operated on for hemorrhage, and of these 2 recovered and 1 died; and finally, 8 cases of peritonitis due to a perforated ulcer of the stomach were operated on, with a mortality of 50 per cent.—4 cases recovered and 4 died. The statistical facts of these few cases do not allow of any particular generalization except to emphatically point out the fact that surgical procedure offers in certain cases the only road to recovery.

THE SURGICAL TREATMENT OF GASTRIC ULCER.*

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There is probably no region of the body at the present day in which there is more surgical interest than the stomach. The recent progress in gastric surgery is largely due to the magnificent work of von Mikulicz, Mayo Robson, Moynihan, and Mayo. These surgeons have had the advantage of attracting and controlling very large amounts of clinical material from which to select their cases. Consequently we have from them many observations upon the treatment of cases which ordinarily do not go to the surgeon, and we have had to, and still, look to them chiefly for advances in the surgical treatment of gastric conditions in general. To make my point more clear it is only necessary to reflect that the surgical treatment of gastric ulcer has been hitherto almost entirely directed to its more serious complications, namely, perforation and cicatricial contraction with its resultant obstructions. It is only in the last few years that hemorrhage from ulcer has been treated, to any extent, surgically; and at the present time we are just beginning to recognize what surgery can do for the chronic ulcer which may give no symptoms beyond slight disturbances of motor sufficiency and indigestion, with occasional exacerbations attended by pain and possible hemorrhage. It is in the latter class of cases that we must look for progress both in diagnosis and treatment, for these cases may proceed to perforation and stenosis for which we know there is no remedy other than surgical intervention. Few surgeons other than those mentioned can report series of operations for ordinary chronic ulcer. For instance, my personal experience includes no such cases but only cases of perforation, pyloric stenosis, or hour-glass contraction and hemorrhage. My hospital service is one of the largest and most active in New York, and the utter absence of cases of ordinary chronic ulcer must be due either to a hesitancy on the part of the general practitioner in that city to resort to surgical measures or to a lack of knowledge as to what surgery can do for such cases.

The treatment of acute gastric ulcer is essentially medical, yet, as has been pointed out by Mayo and others, the exacerbations of a chronic ulcer are probably not infrequently mistaken for an acute ulcer, and the cure is consequently not permanent.

Having stated the aspect of the subject in general, it is my purpose to take up the surgical treatment of the phases of gastric ulcer,

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namely, perforation, cicatricial contraction, and obstruction, hemorrhage, and chronic gastric ulcer, *per se*.

PERFORATION. Unless operated upon, 98 per cent. of cases of perforation die. In 1895 Gould[1] was able to collect only 7 cases that had been successfully operated upon. In 1896 Weir and Foote[2] collected 78 cases, with 29 per cent. of recoveries. In this series the mortality among the cases operated upon within twelve hours after the perforation was 39 per cent., between twelve and twenty-four hours 76 per cent. over twenty-four hours 87 per cent.

Since that time the number of operated cases has gone up into the hundreds, with but little change in the mortality other than the betterment produced by the natural improvement in the treatment of peritonitis caused by perforation.

Although advances in general in the treatment of peritonitis have reduced the mortality, the most important advance has been the establishment of the fact that early operation is absolutely necessary for the attainment of good results. To wait for the shock to disappear is simply to invite failure. The shock of a perforation is caused by irritation of the peritoneum, and disappears with the administration of the anæsthetic. Abundant investigations have shown that the contents of the stomach and duodenum are practically sterile, particularly when hyperacidity is present. In a short time, however, the presence of the foreign material in the peritoneal cavity invokes a violent septic peritonitis.

I have not so far lost a case of gastric or duodenal perforation that has come to me within twenty hours of the perforation. In nearly all of these I have washed out quantities of gastric and duodenal contents which had been scattered throughout the entire cavity, and have closed the peritoneum without drainage.

Advance in the treatment of these cases must be in the line of early diagnosis. The diagnosis of a perforative lesion in the upper part of the abdomen is not so difficult; when once seen the picture can hardly be forgotten. If there is a history of antecedent gastric trouble the diagnosis is simpler; but it must be remembered that ulcers outside of the motor area of the stomach may give no symptoms before perforation. A slow or chronic perforation, as it is termed by Moynihan, often gives rise to a local peritonitis and abscess, and the diagnosis may consequently be obscure; yet in these cases there is not the demand for early interference that pertains to acute perforations.

My personal experience with perforated gastric and duodenal ulcers is limited to 6 cases. Two were in women, the ages being twenty and twenty-one years. In both the ulcer was situated on the anterior surface, one at its middle, the other one-and-one-half inches from the cardia. In one the perforation had existed for six days, in the other twenty-one hours. In both, adhesions had formed which had subsequently ruptured, deluging the cavity

with gastric contents and pus. Both died: one on the tenth day, the other on the sixteenth day after operation, from sepsis from subphrenic abscesses. Abundant drainage was employed in both cases.

The remaining cases were males. One of these, a man of thirty-five years, was operated upon forty-eight hours after the perforation, and had a general peritonitis. The ulcer was situated just to the left of the pyloric ring. Death occurred on the fifth day from persistence of peritonitis. The remaining three cases recovered. One, a man forty-seven years of age, was operated upon eight hours after perforation. The ulcer was situated at the pylorus and could not be closed by inversion; it was therefore excised, the incision being planned so that a more or less typical pyloroplasty was done. The foreign material was flushed out and the abdomen was closed without drainage. Convalescence was uninterrupted. He now, one and one-half years since the operation, has some symptoms of motor insufficiency, and I have recommended gastroenterostomy. Another case was operated upon six hours after perforation. The ulcer was also situated at the pylorus, which was stenotic. The abdomen was full of gastric contents and bile. The perforation was one-eighth of an inch in diameter and was closed by inversion, thus also closing the pylorus. The abdomen was then flushed out and a posterior gastroenterostomy performed. The wound was closed without peritoneal drainage. Recovery was rapid. The last case was a man twenty-five years of age, with a small perforation of the duodenum close to the pyloric ring, of forty-eight hours' duration. There was a well-established general peritonitis, with marked distention. A diagnosis of appendicitis was made, with a corresponding incision. Afterward a median incision was made, the perforation found and closed, the ileum opened and emptied to permit of reduction of the intestines that had escaped, and again closed; the abdomen thoroughly flushed and drained by a stab wound below the right ribs and by a drain in the pelvis through the first incision. Recovery was somewhat slow but complete.

Recovery in the cases in this series operated upon within twelve hours was rapid and exceedingly satisfactory. The same has been true of a number of traumatic perforations of the stomach and upper intestinal tract that I have operated upon.

Cicatricial Contractions and Obstruction. Obstruction from cicatricial contraction occurs most often at the pylorus, more rarely at other portions, where it gives rise to hour-glass contraction. Lesser grades of interference with motility are caused by perigastric adhesions or even by the mere presence of ulcers without obstruction in the pyloric portion, and will be referred to later in connection with chronic ulcer.

Pyloric and hour-glass obstructions can be helped only by surgical intervention, which gives most excellent results, the mor-

tality in these cases, unless the patient is nearly moribund from inanition, being extremely low, in the neighborhood of 5 per cent.

While in the cases of uncomplicated pyloric obstruction, Finney's operation has been productive of excellent results, in stenosis due to ulcer gastroenterostomy, with or without pylorectomy, appears to be the operation of choice. Apropos of gastroenterostomy, I venture to report one of my cases of hour-glass stomach somewhat fully, since nature had already elected a posterior gastroenterostomy as a solution of the difficulty. In this patient, a woman twenty-six years of age, I found a typical hour-glass stomach, the constriction being at its middle. On turning up the colon I found what was apparently a congenital deficiency in the transverse mesocolon through which a loop of the ileum had passed and become adherent to the posterior wall of the stomach at the site of the contraction. In liberating this loop it became apparent that the ulcer had penetrated through the coats of the stomach and partially through those of the small intestine. In closing the aperture in the stomach I was able to enlarge the constriction and then perform a gastrojejunostomy with the cardiac pouch. This patient has entirely recovered her health and has no gastric symptoms.

HEMORRHAGE. Hemorrhage from gastric ulcer is a serious complication. Savariand[3] places the mortality at 66 per cent. In regard to the surgical treatment there are many differences in opinion. The statistics gathered from the reports of isolated cases are also misleading. These variations are traceable primarily to the numerous phases of hemorrhage occurring from ulcer or, perhaps, to put it better, to the varieties of ulcers which happen to bleed.

We owe to Moynihan[4] the most succinct exposition of the subject of hemorrhage from ulcer. His division of the cases is dependent upon whether the hemorrhage occurs from an acute or chronic ulcer. Hemorrhage from an acute ulcer, although frequently violent and depleting, displays a marked tendency to subsidence under medical treatment. This explains the dictum of other observers, Leube,[5] Hartmann,[6] Lund,[7] and others, that operations should not be done for single large hemorrhages. On the other hand, hemorrhage from a chronic ulcer is usually repeated. The hemorrhages may be frequent and small, resulting only in a persistent anæmia, or they may be large and recur at long intervals, or be rapidly repeated. Again, we may find that the hemorrhage may come from one or many small superficial erosions or from one or several typical chronic ulcers.

These variations in the origin of the hemorrhage constitute therefore the differences in the indications for surgical intervention and in the technique to be employed.

My personal experience has been limited to 5 cases operated upon for hemorrhage, with two deaths. Both the fatal cases had

chronic ulcer from which there were several rapidly repeated large hemorrhages. In one the percentage of hæmoglobin was 35 per cent. before operation, in the other 33 per cent. In the first case the ulcer, which was at the middle of the anterior surface of the stomach, was excised, no gastroenterostomy being done. In the second case gastroenterostomy alone was performed. The first case died on the sixth day from what I considered meddlesome treatment in the shape of large repeated infusions, by which the percentage of hæmoglobin was reduced to below 20. The second case died from an extension of the thrombus in the bleeding vessel back into the hepatic artery. In this case the ulcer was situated in the middle of the lesser curvature and had been practically latent. In one of the three cases that recovered a very small round ulcer was excised from the middle of the posterior wall, no gastroenterostomy being done; in the other two, gastroenterostomy alone was performed, the ulcer not being located. These cases had had several fair-sized interval hemorrhages, but were in excellent condition. There has been no recurrence of hemorrhages since the operations. In two of them, however, only three months has elapsed.

My opinion is, that a single large hemorrhage, without previous symptoms referable to ulcer, should not be operated upon, but when there have been antecedent symptoms, operation should be at once performed. In no case should the patient be allowed to drift into a condition of profound anæmia from successive hemorrhages. Also, that cases suffering from recurrent hemorrhages, whether small or large, should be operated upon.

In regard to the nature of the operation I believe that excision of the ulcer combined with gastroenterostomy is the method of choice. Excision, however, is out of the question in profound anæmia and when the ulcers or points of hemorrhage are multiple. It should be done, if practicable, when the ulcer is not in the pyloric portion. Ulcers in the pyloric portion are probably much more amenable to gastroenterostomy alone than when situated elsewhere. Moynihan is a firm believer in the efficacy of gastroenterostomy alone.

Chronic Gastric Ulcer. As already mentioned, the treatment of acute gastric ulcer is essentially medical, since in these cases the results are extremely good. The story in chronic ulcer is different. Russell's[8] statistics derived from a large number of outpatients are that 42.6 per cent. recovered; but of these 27.7 per cent. had had only one attack, and should therefore be classed as acute ulcers, thus leaving only 14.9 per cent. which recovered after having more than one attack. The remaining cases, excepting those that died, were all suffering from gastric symptoms of more or less severity. The average of the figures given by von Mikulicz,[9] Mayo Robson,[10] and others who have collected statistics is not far from

25 per cent. mortality if the deaths from tuberculosis, etc., to which these patients are predisposed, are not included. With these the figures approach 50 per cent. In 1897 von Mikulicz,[9] in a series of 238 operated cases from the clinics of Billroth, Czerny, and himself, found the mortality to be 16.1 per cent. In 1900 Mayo Robson,[10] in 188 collected cases of operations for all complications (excluding perforation and hemorrhage), found the mortality to be 16.4 per cent.; yet in 1901 he reports the mortality of his own cases to be only 5 per cent. Moynihan[4] reports 88 similar cases with a mortality of less than 5 per cent. The Mayos'[11] statistics up to April 1, 1904, in 328 operations for the improvement of gastric drainage, some of which were performed for malignant diseases, are as follows: 20 pyloroplasties, no deaths, but seven secondary operations; 40 Finney gastroduodenostomies, one death; 271 gastrojejunostomies, with a mortality of 6 per cent. in the benign cases. In the very few cases which I have operated upon for benign pyloric or other obstructions I have had no deaths.

It may be well to consider the reasons, other than a distinctly lesser mortality, why surgical treatment has the advantage. Besides the elimination through excision of the tendency to malignant degeneration, which occurs in 6 per cent. of the cases, there are still other points. In the first place it is a question whether the hyperchlorhydria which often accompanies ulcer is not a result instead of a cause of the ulcer. Soupault[14] reports complete relief from hyperchlorhydria and gastrosuccorrhœa in several cases in which pylorectomy was done for ulcer at the pylorus. Pylorospasm, which is frequently caused by the presence of an ulcer, at once yields to excision or gastroenterostomy.

The majority of symptoms causing ulcers are situated in the pyloric portion and are consequently at a point where the utmost benefits are derived from gastroenterostomy. On the other hand, let us for a moment view the matter from the opposite standpoint and consider the dangers of gastroenterostomy. The operative procedure to one who is accustomed to it is very simple and is followed by very little discomfort or shock. I am always surprised at the slight reaction following the operation, it being usually much less than that following the interval operation for appendicitis. The relief, if pyloric obstruction has been present, is immediate, and is evidenced by the general expression of the patient even on the day following the operation.

The vicious circle, owing to better methods, is no longer to be feared. It was due apparently to the employment of a long loop of intestine in either the anterior or posterior operation with a too high implantation in the stomach. It has been combated by an enteroenterostomy, by the Roux method, by a lower implantation, and, finally, by eliminating the loop and using the very commencement of the jejunum where it naturally lies in relation with

the posterior wall of the stomach, as in the Petersen operation. I have found that this method gives excellent results.

Peptic ulcers in the jejunum sometimes follow gastroenterostomy. Mikulicz,[12] in a study of 15 cases, finds that they are not dependent upon a hyperchlorhydria as is usually supposed, but that they may occur when the percentage of hydrochloric acid is below normal. He also finds that they occur only after anterior gastroenterostomy. This may be explained by the fact that a much longer loop of jejunum is necessary in the anterior operation and that consequently the mucous membrane is less protected by the bile.

The danger of contraction of the anastomotic opening is largely preventable by a proper technique which eliminates the production of excessive scar tissue.

Many ask, "Why does gastroenterostomy cure gastric ulcer?" The explanation of its curative action upon ulcers situated in the pyloric portion is simple. This part of the stomach is very muscular, and its chief function is to force the partially digested food through the pylorus. Consequently an ulcer situated there is constantly subjected to traumatism, not only by the contractions of the stomach, but also by the passage of food across it. This trauma is accentuated if there is any obstruction, organic or spasmodic, at the pyloric opening. A proper drainage opening situated to the left of the pyloric portion at the most dependent part of the stomach affords complete rest to the pyloric portion and at the same time drains away any excess of gastric secretion.

Its beneficial action upon ulcers in the cardiac portion is not so clear, but here, also, the more rapid emptying of food and secretions must produce some result. If possible, however, it seems to me that excision of ulcers in the cardia should be practised.

But besides the institution of drainage there is a further field for surgery in the liberation of adhesions, the plication of dilated stomachs, and in fixation.

Viewing the results surgery has accomplished in these cases, one must concede that the treatment of gastric ulcer should be, at least, largely surgical. Our plea is for earlier resort to surgical measures; not only in ulcer but in malignant disease. By the time the diagnosis is evident by chemical or physical aids the patient is often beyond relief. As Munro[13] puts it, surgical treatment should be instituted "not late but early; not as a last resort, but before the starvation, the long suffering from pain, dyspepsia, and fermentation absorption have reduced the patient until he is beyond surgical help; before he is anæmic from persistent bleeding or peritonitic from culpable delay."

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A CASE OF GENERAL MILIARY TUBERCULOSIS, WITH AN OPEN FORAMEN OVALE AS A FACTOR IN ITS PRODUCTION.

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The role of a persistent foramen ovale in permitting pieces of thrombi and tumor cells to pass directly from the right auricle to the left side of the heart has been brought into prominence by various investigators. In none of the literature consulted is there mention of the passage of bacteria through such an opening in the interauricular wall. It is our opinion that we have a case in which tubercle bacilli were disseminated through the avenue of an open foramen ovale. Other interesting features of the case now to be described are the extent and size of the tuberculous nodules on the peritoneum and the tuberculous invasion of the pancreas. Though aware of the hypothetical nature of the conclusion regarding the pathogenesis, I feel, from the data, that the inference is highly probable and of great interest in relation to general miliary tuberculosis. Throughout the work I have had the assistance and helpful criticisms of Dr. A. P. Ohlmacher, the director of the pathological laboratory of the Ohio Hospital for Epileptics.

Clinical History. Only the following points have been obtainable: C. E., aged twenty-four years, was a male, colored, an imbecile, and an inmate of the Ohio Hospital for Epileptics for ten years. He had had idiopathic epilepsy for thirteen years. His grand mal attacks occurred principally at night and at irregular periods. His family and previous history were unobtainable, except that until the day before his death he was up and about and that within the last two months of his life he had failed visibly in weight and strength. His physician was called one evening for the reason that during an attack the patient had bitten his tongue severely, almost severing the organ about an inch from the tip. Examina-

tion revealed a startling prostration, a temperature of 101°, a weak, irregular pulse of 127, and a respiratory rate of 40 per minute. That the tongue wound could cause the observed signs was not deemed possible, but before enough data could be collected for a diagnosis the patient died. The lethal ending was not attended with status epilepticus nor with any single grand mal attacks.

Gross and Microscopic Morbid Anatomy. The body was fairly well nourished. The anterior inch of the tongue was almost entirely separated from the rest by a deep, gangrenous wound. The oral cavity was otherwise normal. Subcutaneous lymph nodes, except the inguinal, were not palpable, and the latter showed no unusual size. The testicles were of equal size, small, and soft, and presented no nodules to the touch. There was no fluid in the abdominal cavity. The parietal peritoneum adhered to the omentum. The intestines were matted together. All these adhesions could be broken without much difficulty. Both surfaces of the abdominal serosa were studded with an immense number of large, yellowish, confluent, elevated nodules, which were somewhat hard and recalled to mind tumor metastases. The liver and spleen were likewise somewhat firmly bound by the peritoneal exudate to the diaphragm, to the parietal peritoneum, and to the neighboring viscera. The pancreas was practically embedded in the exudate and in a cordon of large glands. The mesenteric lymph nodes were only slightly enlarged. The iliac, the lumbar, the aortic, and especially the cœliac glands were much increased in volume. The anterior and posterior mediastinal glands were much above normal size. There were adhesions in each pleural cavity over the entire lung, which connections were easily broken by the hand. There was no fluid in either pleural sac.

The heart and pericardium together weighed 425 grams. The latter was adherent to the heart all over. On stripping off the pericardium some yellowish nodules were seen. The cardiac orifices and valves were normal. There was a slight hypertrophy of the left ventricle. The musculature of the right auricle was thickened and its endocardium was cloudy and dense. The foramen ovale was open, and a diagonal slit, admitting a one-quarter inch probe and running anteroposteriorly from left to right, made communication between the two auricles. The fossa ovalis was deep and bulged to the left. The ring of Vieussen was well developed and protruding, and was continuous with a thick, pronounced Eustachian valve. Microscopic examination revealed fresh tubercles in the thin exudate on the epicardium. The musculature showed a recent myocarditis, but no tubercles after a search through many sections.

The right lung weighed 930 grams, the left 800 grams. They were similar, of a deep-red color, and crepitated somewhat. The pleura was slightly thickened. Cut surface showed a pale brownish

color and numberless whitish tubercles of the size of millet-seeds, which were likewise plainly seen when the pleura was stripped off. Both lungs were searched for old caseated foci, but none was found. At the apices the tubercles were somewhat larger to the eye than in the rest of the lung, a fact which is of interest in view of Hansemann's[1] recent contention that in simple miliary tuberculosis there is no difference; but there were no old lesions, like cavities, fibrous changes, or calcareous deposits. The bronchial glands were large, but were discrete and not soft. Microscopic examination of the lung itself showed many tubercles of small size, in the largest of which there was some breaking down in the centre, but practically all of them were discrete. The lymphoid and epithelioid elements of the tubercles were distinct, but it is noteworthy that giant cells were found in only very few of them. Other characteristics of the lung sections were a moderate dilatation of blood-vessels and many alveoli and bronchi filled with cells, a mucoid substance, and considerable detritus—in short, some of the evidences of bronchopneumonia.

The spleen weighed 440 grams, and was adherent to the diaphragm, the liver, the stomach, and the intestines. At the hilus of the organ there were four or five somewhat enlarged lymph glands. The spleen was large and firm, and, on the capsule, there were numerous large, yellowish, elevated nodules, similar to those on the other peritoneal surfaces. It cut readily and the brown pulp showed no increase of connective tissue but a great abundance of whitish, pinpoint spots universally diffused. Microscopically it was somewhat difficult to differentiate the miliary tubercles at first glance, inasmuch as there were no giant cells present and the Malpighian bodies were numerous. However, on closer study, the epithelioid cell centre and ring of lymphoid cells served to distinguish the tubercles from the Malpighian bodies with their uniform structure of lymphoid cells and the accompanying arteriole. Only here and there were tubercles showing a central degeneration.

The liver weighed 1840 grams, and was bound firmly to a mass formed by the pancreas, the stomach, neighboring parts of the large and small intestines, and some large lymph glands near the cœliac axis, one of which was almost as large as a hen's egg and situated a little to the left of the hilus of the liver, closely associated with the portal vessels. The gall-bladder was in the mass, and one could not say whether the ducts were patent or not, for, owing to these adhesions, the bile could not be squeezed out nor could the ducts be probed. There were no gallstones in the gall-bladder or ducts. The liver was large and firm. There were nodules on its capsule similar to those on the spleen. The organ cut readily and the incised surface showed many whitish tubercles of pinpoint to pinhead size. The markings of the liver were indistinct and there was no congestion. Besides its involvement with

the structures in the mass above mentioned, the liver was adherent to the diaphragm, to the spleen, and to the right kidney. In stained sections the liver showed a great number of fresh tubercles; in fact there were more in the liver in any microscopic field than in the lungs. Here, again, it was noted that only a few presented well-formed giant cells, although in the liver sections the greatest number was found. The tubercles were all of approximately the same structure, with practically no central caseation. On the whole the age of the tubercles, judged from their microscopic appearances, must have been about the same in the liver as in the lungs and spleen. The hepatic parenchyma was otherwise about normal. The intercellular connective tissue was prominent, but there was nothing approaching a cirrhosis. There was no abnormal distention of the vascular system.

The left kidney weighed 165 grams. It was large and waxy looking. The capsule stripped with ease and did not tear the parenchyma. The decapsulated surface showed only a slight injection and a few whitish, circumscribed tubercles, some of which were seen on cut section. The cortex was somewhat increased in thickness. The right kidney weighed 150 grams and was similar to its fellow except that on cut section tubercles could be made out more distinctly. Microscopically a few fresh tubercles were seen in the cortex and in the medulla of both kidneys. Here none was found with the typical giant cells of tubercle, although in one there was a large cell with several nuclei. Otherwise the following conditions were found: (*a*) glomerular tufts were much distended; Bowman's capsules were unchanged, but between the tuft and the capsule there was a good deal of detritus, with an occasional cell; (*b*) the epithelium of the renal tubules was cloudy and desquamated; (*c*) considerable vascular dilatation; (*d*) connective tissue was not increased—briefly the changes of an acute diffuse nephritis. Sections were treated for the amyloid reaction, with a negative result.

Both suprarenal capsules were small and hard, but showed no nodules or caseation. Microscopically, besides the usual fatty infiltration, there was noted a pronounced hyperæmia, especially toward the medulla. There was no degeneration of cells. After examining a considerable number of sections, two tubercles were found, one fresh and one with a partial central degeneration, and neither showing a giant cell.

The pancreas weighed 120 grams, and, as mentioned before, was surrounded by enlarged tuberculous glands, and directly upon its head lay the largest of the cœliac glands. Microscopically there was no increase of connective tissue, the gland tubules were unchanged, and the islands of Langerhans were intact and of usual size. One partly caseated tubercle was found in the parenchyma in the lower border and just at the edge. It was entirely within the pancreas, though its outer lymphoid ring extended to the

extreme edge of the lower border of the organ. A more prolonged search would probably have demonstrated other tubercles.

The prostate was soft and not enlarged, and microscopic examination disclosed no trace of tuberculosis or other inflammation. The testicles were, by an oversight, not saved, but palpation had revealed no nodules or enlargements of any kind, and both vasa deferentia were, at autopsy, found regular and of their usual consistency.

The peritoneum, as indicated before, exhibited a most striking hyperplastic tuberculosis. The nodules were as large as a bean in many locations, and the whole process was diffuse and confluent. The tumor-like nodules reminded one a good deal of the "Perlsucht" of cattle, and compared favorably with the description of a form of latent tuberculosis of the peritoneum given by Kaufmann.[2] Sections of such nodules showed an extensive caseation, only the periphery containing tissue not degenerated. There were no ulcers in the intestines, which were negative except for a slight increase in lymphoid tissue.

All of the abdominal and thoracic lymph glands presented a great augmentation in size, but a gradual decrease in size of the glands from the abdomen up was noticeable. The largest ones were the lumbar and cœliac. Those in the mediastinum were about the same volume as the bronchial glands. The deep cervical nodes were slightly larger than normal. Unfortunately, nothing further can be said of the inguinal glands except that through the skin they showed no marked increase in size. Microscopically these all revealed an advanced caseation. Those in the cervical region were similarly destroyed, but in them the process could be denoted comparatively recent, for there were many fresh, well-developed tubercles present.

There was a persistent thymus, with imperfectly developed corpuscles of Hassal, but no tuberculosis of that organ was found, though it lay within a mass of tuberculous glands. The thyroid was negative.

The brain weighed 1235 grams. The dura was unchanged. The pia was slightly cloudy over the convexity. Otherwise the encephalon was negative except that there were a few slender adhesions between pia and dura at the base in the region of the circle of Willis and under the frontal lobes. No tubercles could be definitely made out, and, in spite of the basal adhesions, sections of the pia in those regions contained no evidence of tuberculosis. The spinal cord was negative.

Direct smears from the peritoneum and liver showed no acid-resisting bacilli. In those from the lungs and spleen only a few such bacteria were found. Blood-serum inoculations from the spleen, heart's blood, peritoneum, and pia remained sterile. From the lung staphylococcus aureus and albus were isolated. From the liver there was obtained a short, thick bacillus, which reacted

in all ways like the colon bacillus, and was probably an artificial contamination.

COMMENTS. In summing up and interpreting the foregoing data, I wish to prove three things: first, that the primary seat of the affection was in the abdominal cavity; second, that the general tuberculosis originated by the lymphogenous route; third, that the open foramen ovale was instrumental in the production of the generalized disease.

It is of prime importance to take some position regarding the original seat of the inflammation. The tuberculosis of the lungs, the liver, the spleen, the kidneys, the suprarenals, the pancreas, and the pericardium was of recent origin, while that of the thoracic and abdominal lymph glands and of the peritoneum was, judging from the size of the nodules and from the extent of the caseation, much older. For these reasons, I assume, and I believe with justification, that the tuberculosis began in the abdomen, and there, no doubt, in the peritoneum, inasmuch as such an affection is not uncommonly primary in the serous covering.

Taking this for granted, under our second heading, the chain of lymph glands from the iliacs up demands some consideration. The lymph nodes around the iliac vessels were considerably larger than usual, the lumbar were still larger, and the cœliac glands were most extreme in size. This increase in volume corresponds with the areas drained by, and with the tributaries of, the groups mentioned. Following up through the thoracic cavity the mediastinal glands were all enlarged, about the size of those in the lumbar region. This chain of glands, I believe, was the path by which the tuberculous material travelled from the peritoneum to the thoracic duct and thence to the heart. This is one of the acknowledged paths for the production of a general miliary tuberculosis, it making no essential difference, as far as result is concerned, whether the tuberculous material breaks through the walls of the thoracic duct or arrives there from its tributaries. This route, first demonstrated by Ponfick, is found especially in tuberculosis of the glands of the abdomen.[3] The thoracic duct was not discoverable in the mass of tuberculous tissue, but the fact that we did not find it is of no importance, for, from the anatomical relations of that vessel to the general glandular system, it was destined to transmit the infectious material.

The possibility of a miliary tuberculosis, consequent to the invasion of a pulmonary bloodvessel, was carefully excluded. Beginning with the arterial and venous trunks, all of the bloodvessels of the lungs down to the smaller ones were carefully slit open in search for a thrombus, an embolus, or an eroded vessel. The result of this dissection was entirely negative. To put this conclusion beyond criticism, sections were cut from the areas where the bronchial glands lay nearest the intima. This examination removed any

possible doubt. Nowhere did the tuberculous process approach nearer than the adventitia of the vessel, and, in the majority of cases, a dense connective-tissue ring interposed between the outer coat of the vessel and the degenerative process.

Finally, it is necessary to support our third contention. Having arrived at the right side of the heart, there are possible, because of the presence of the open foramen, two distinct means of dissemination of the tuberculous material. One is by way of the pulmonary artery to the lungs and then back to the left heart and thus to the general circulation, constituting a condition of secondary emboli. Against the assumption that this was the exclusive path, the appended arguments could be brought: (*a*) The lungs in our case do not show any greater invasion than the liver or spleen, which they would have done under the above hypothesis, for tuberculosis is pre-eminently an embolic process.[4] That conditions for embolism and thrombosis are present in capillaries only a few hours after tubercle bacilli arrive in them has been shown recently by Wechsberg[5] and by Miller.[6] All of the organs involved in the acute distribution of the bacilli in our case showed tubercles of about the same age, and though some of the bacteria could undoubtedly escape the lungs and get to the systemic circulation, as careful microscopic work would show, there would not be present, under those conditions, the uniform number of tubercles of the same structure as were found in the lungs, liver, and spleen. (*b*) There would not be present such auspicious conditions in the heart, the details of which have been enumerated and will be further described. (*c*) Such a pulmonary route would probably not be the one in a rapid development of the disease as occurred in our case, evidenced by the lack of giant cells, as indicated by Birch-Hirschfeld,[7] and by the nature of the history of the case. (*d*) Such a path is operative in all cases where tubercle bacilli pass through the pulmonary artery to the lungs, causing miliary tuberculosis there, and yet a systemic invasion of equal grade in all organs is not shown in such instances. The literature has been searched in vain for some specific statement upon the question, not which organs are affected in hæmatogenous miliary tuberculosis when the embolic material passes first to the lungs from the right heart, but whether the intensity and age of the process in the several organs are equal. The table given by Eichhorst[4] (p. 614) and the ones quoted by Birch-Hirschfeld[7] (p. 351) are not to the point. Recently, however, some experiments have been performed by de Haan[8] which give an answer to this question. Assuming, with probability, that there was no open foramen ovale in either of two animals inoculated by de Haan, and to be mentioned here, the conditions of these experiments are just those that I am now discussing and am trying to show to be inadequate to have caused the general miliary tuberculosis of our case. De Haan inoculated tubercle bacilli into the

left jugular vein of a horse and of a Javanese beef. At the end of seven weeks, because of great prostration, the horse was killed. The autopsy showed the liver, the spleen, the pericardial sac, and the abdominal cavity to be normal. There were a few tubercles in the kidneys. The lungs, however, were so invaded by tubercles that there was present almost no air-containing tissue. The other animal was killed at the end of nine weeks. It had been affected with the distoma, and the liver presented a corresponding change, but no tuberculosis of that organ is mentioned. The lungs were much involved with miliary tubercles, but the other organs were normal. To the argument that a single introduction of tubercle bacilli invalidates these results for our purpose, it may be stated that in the experiment with the horse a cheesy thrombus remained at the sight of inoculation, which was always a source of further infection. An added proof, the value of which may be questioned, is the work of Strauss and Gamaleia,[9] who injected intravenously a culture of dead tubercle bacilli and found miliary tuberculosis of the lungs, but none of the spleen or liver. (*e*) It appears, from anatomical relations and from the hyperplastic nature of tuberculosis, that the infectious material must get directly into the aortic side to cause a widespread miliary disease and of equal magnitude. Because of all these enumerated facts and arguments, I feel confident that the general miliary tuberculosis of our case was not produced by way of the pulmonary artery. For the sake of completeness, it might be mentioned that a very improbable explanation of the miliary disease under discussion is by retrograde lymph flow to the organs from the glands into which they drain. It is highly probable that the cervical and, no doubt, the inguinal glands were invaded this way from the thoracic nodes. The age of the process in the former glands is too extreme to have been produced by the route that caused the tuberculosis of the other organs.

The other method of spread and the only other avenue open is by way of the patent foramen ovale. The conditions in our case are exactly those that Rostan[10] and Zahn,[11] who have thoroughly discussed the subject of embolism through an open foramen ovale, call the essentials: a thickened endocardium and an increase in the thickness of the musculature of the right auricle; a well-developed Eustachian valve; thickened and protruding borders of the fossa ovalis; a bulging to the left of the base of the fossa; and an open foramen, which in our specimen admits a quarter-inch probe. The cause that will produce an increase of pressure in the right auricle, so imperatively demanded before there will be a passage through the foramen, was certainly present as a bronchitis, and later as a bronchopneumonia at the first onslaught upon the lungs by the bacilli by way of the pulmonary artery. Rostan says that the bulging of the valve of the opening is a positive proof of

such an augmentation of pressure, and Zahn states that, besides this bulging of the fossa to the left, the best anatomical proofs in the right heart of the presence of a stasis and an increase of pressure are the hypertrophy of the muscular walls, the enlargement of the fossa, and the thickening of its edges. Therefore, we have all the requisites for the passage of blood and foreign material from the right auricle to the left, and, though the tuberculosis of the lungs was in large part produced by way of the pulmonary artery, it is highly probable, with such a direct path open, that the production of the general miliary tuberculosis occurred in largest part by way of the open foramen ovale to the aortic side. With the large percentage of such persistent openings in the interauricular wall that is found at autopsy (v. Rostan and Zahn, *loc. cit.*), it is surprising that this route has not been dwelt upon before.

The tuberculosis of the pancreas in our case is of interest only because of its apparent rarity. Most writers dismiss the subject with the statement that it is not often seen. According to some authors, when such a condition is found it exists in a lymph gland on the surface or embedded within the pancreas. Kundrewetzky,[12] however, in 128 cases of consecutive tuberculosis, found 12 positive tuberculous affections of the pancreas, or 9.37 per cent.; in 18 cases of general miliary tuberculosis he found this organ invaded six times, or 33.3 per cent. In children he found 44.44 per cent.

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A BICEPHALOUS MONSTER.

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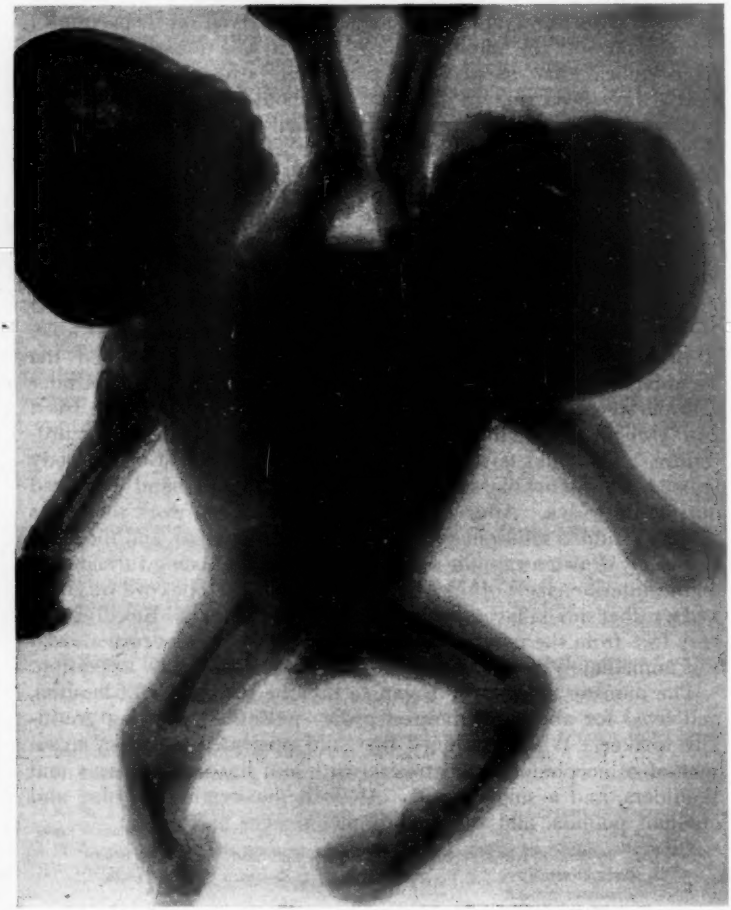
On March 21, 1904, a young woman was admitted to the hospital in labor at term. Nothing unusual was observed on inspection of the contour of the abdomen save that it was probably slightly more conical than usual. The pains were strong and normal in every way, occurring at intervals of about three minutes. On vaginal examination an apparently normal L. O. A. presentation was diagnosed; membranes intact and well down on the pelvic floor. These were ruptured, and the scalp speedily appeared at the vulvar opening. The delivery of the head was more tedious than was expected, retraction after each pain being very marked. It was finally accomplished, however, but rotation did not follow, nor could the child be drawn down. After a short interval the head turned rather rapidly toward the left, rotating about 90 degrees. Here it stopped, and the child seemed absolutely immovable. Efforts at traction were unavailing. Examination showed the shoulders blocking up the pelvic outlet and tightly wedged. Under full anæsthesia, by manipulation and traction, the arms were freed and brought down, though both humeri were broken in the procedure. The child now began to breathe, but the rest of the body seemed as immovable as ever. Suddenly a third hand appeared outside the vulva. After about half an hour's hard work there was a second sudden rotation, still to the left, of 90 degrees, and the child was easily drawn away, the second and right head being turned over on the anterior aspect of the thorax facing the left. The cord was tied, and a rather small placenta delivered without difficulty. Bleeding was very free from the cervix, which showed a bilateral laceration, and was immediately closed by sutures. There was no external laceration.

The monster after birth began to breathe through both mouths, and lived for about two hours, cardiac pulsations growing gradually weaker. When examined the child presented a perfect specimen of a bicephalous monstrosity, with two heads, four arms and shoulders, and a single trunk. At birth the weight was nine and one-half pounds, and the measurements were as follows:

	Left head.	Right head.
Circumference	33 c.m.	33½ c.m.
Diameter of heads:		
Bitemporal	7 "	7 "
Biparietal	8 "	8¼ "
Occipitofrontal	11½ "	10 "
Occipitomental	12 "	12½ "
Suboccipitobregmatic	12 "	13 "

Circumference of thorax	46 c.m.
Diameter of thorax, sagittal	7 "
Parietal	20 "

The fusion anteriorly seemed to be complete below what normally would have been the junction of the manubrium and gladiolus, while posteriorly there were two distinct vertebral columns, the inner set of ribs fusing by cartilaginous bands. The pelvic girdle consisted of five distinct bones—two normal external ilia,



joined anteriorly in a normal symphysis; two normal sacra, each with an attached coccyx, and a wedge-shaped bone uniting the sacra and evidently representing the fusion of the internal ilia. The details of the bony skeleton are well shown in the accompanying skiagram prepared by Dr. S. H. Heller.

After the death of the monster a partial autopsy was made, care being taken not to injure it more than absolutely necessary. The abdominal cavity was opened, and the contents appeared nearly normal. The anus was imperforate, and the rectum enormously distended with meconium. There was a single stomach, into which two œsophagi entered in the median line through practically a common opening. The liver was large, and apparently from its irregular shape represented the fusion of two organs, there being, however, but a single gall-bladder. The kidneys were normal, the testicles undescended. On examining the thorax two hearts were found in a single pericardial sac, while the lungs showed two apparently normal external lobes, while the internal lobes were more or less fused.

The mother of the child was of German extraction, belonging to one of the numerous religious sects which make up the farming population of Lancaster county. She was eighteen years of age, unmarried, and had been delivered late in March, 1903, of her first child. She was the oldest of a large family, and nothing abnormal could be ascertained in her family history. The father was a light mulatto, and the child showed unmistakable evidence of negro blood. The mother's convalescence was uneventful.

TWO CASES OF VIOLENT BUT TRANSITORY MYOKYMIA AND MYOTONIA APPARENTLY DUE TO EXCESSIVE HOT WEATHER.

By David L. Edsall, M.D.,
of Philadelphia.

Localized acute muscle cramps, especially in the calves, are not uncommon and are sometimes very severe; but, excluding tetanus and other conditions due to poisons or to distinct lesions of the nervous system, widespread cramps are rare, and, as acute conditions, they are seen chiefly in connection with profuse diarrhœa (cholera and cholera nostras) or tetany; and even in these conditions the spasm does not usually involve the trunk muscles. Furthermore, true tetany is seldom and cholera almost never met with in this country.

In the cases that I am about to describe there was exceedingly violent tonic spasm, involving all the extremities as well as the abdominal muscles; the characteristics of tetany were not present; there was no noteworthy digestive disturbance; and the tonic spasms occurred as interruptions of a most remarkable grade of myokymia. The whole disturbance subsided with extreme rapidity,

and in both cases was apparently due to exposure to excessive heat. The myokymia was an even more peculiar and striking feature of the cases than the tonic spasm. Very high grades of fibrillary contraction are, at best, rare, when unassociated with demonstrable organic disease; and I cannot determine that myokymia of such brief duration as that in my cases, but, at the same time, of a comparable degree of severity, has heretofore been described.

In their chief symptoms my cases resembled, on the one hand, the frequently quoted cases of Talma, which he thought presented conditions so far resembling those in Thomsen's disease as to deserve the name of myotonia acquisita; and, on the other hand, the cases of Kny and Schultze, in which there was a remarkable grade of myokymia without noteworthy tonic spasm. As Schultze remarks, the chief feature in his case was the severe fibrillary contractions; while in Talma's the chief feature was tonic intention spasm; in my cases, however, both these features were present and were very severe; and the most remarkable fact concerning them was their brief duration.

Besides the brevity of the attacks, their relation to heat is of interest. In the cases of Kny and Schultze, and in two of Talma's, the conditions present were referred to exposure to wet and cold; and others have made similar observations. The frequency of muscular cramps in swimmers, too, is a matter of tradition. I think that slight degrees of localized cramp to which the subjects pay but little attention, and which occur particularly in the calves, are not uncommon during excessively hot weather, even when digestive disturbance is absent; but I do not know that exposure to excessive heat is recognized as a cause of myospasm. The literature on sunstroke and heat exhaustion, at any rate, contains, so far as I can determine, no reference to such a complication or sequel.

In this relation it is interesting that one of my patients was so subject to cramps when bathing that he had been unable to learn to swim; and, on the other hand, that before the severe attack during which I saw him he had repeatedly had mild attacks of cramps in the extremities when overheated, and when no digestive disturbance was present. Evidently there was in this case, as shown by the history of the patient's childhood, a persistent tendency to myospasm; and, with this predisposition, extremes of either heat or cold tended to cause more or less severe myospasm, his attack while under my observation having been a great exaggeration of the more common conditions.

Although this man was admitted after the other, I shall describe his case first, for I did not see the other patient during his most acute symptoms.

CASE I.—J. H. P., aged thirty-one years, a locomotive fireman, was admitted to the Episcopal Hospital July 3, 1903. His family history was entirely without bearing upon his condition; of his

immediate family, he had lost one brother, of an unknown cause. He had had measles and scarlet fever during early childhood, and pneumonia at the age of nine years, and was said to have had walking typhoid fever later. He had malaria three years ago, having had this disease also when a child. He is subject to brief attacks of diarrhœa, which he thinks are largely due to drinking ice-water. During the last year, when overheated, he repeatedly had in his hands and feet cramps somewhat resembling those in the present attack, though very slight in degree. He has also never been able to swim on account of his tendency to these cramps. He states that upon emotional or other excitement he has always had a good deal of twitching in his hands, and that when at school, if told to write upon the blackboard, excitement sometimes increased this twitching to such an extent that he was unable to hold the crayon and would repeatedly drop it. He has also often noticed slight twitching in the calf muscles.

The present attack began on July 2, 1903, at about three o'clock in the afternoon, while the patient was at work on his engine. The weather was very hot; he became greatly overheated, and was suddenly seized with painful cramps, beginning in the fingers, and almost immediately afterward appearing in the calves of the legs, the thighs, the forearms, and the abdomen. These cramps gradually grew worse until the time of his admission to the hospital. For some hours before admission they had been very frequent and exceedingly painful, although the individual spasms were of brief duration. He had had no disturbance of digestion at the beginning of the attack or until about fifteen hours thereafter. He then became somewhat nauseated and vomited once. His bowels had been normal. He had eaten a small piece of salt mackerel on the morning of July 2d; but, except for this, he knew of no possible dietetic cause. He had taken no other sea-food, no fruit, and no milk or ice-cream.

Upon admission, physical examination showed the patient to be a muscular, well-nourished man. His face was flushed, as the result of his occupation and of the slight fever present. His tongue was clean, but rather dry. His abdomen was neither distended nor tympanitic, and there was no abdominal discomfort other than that caused by the frequent spasms in the muscles of the abdominal wall. The lungs were clear throughout, and the heart was negative. The arteries were not at all sclerosed, so far as could be determined. Examination of the liver, the spleen, and other abdominal organs was likewise entirely negative. There was slight enlargement of a few lymph glands in the groin and the axilla, and a few very small cervical glands could be felt.

The most striking feature of the case—and a very remarkable one—was the constant fascicular contraction in the muscles of the feet, legs, thighs, abdomen, hands, and forearms. There were

also occasional slight twitchings of the muscles of the nose and of the rest of the face, and of the upper arm. This movement was by far most pronounced in the muscles of the calf, where it gave rise to continuous pulsatile or wave-like motions beneath the skin, these taking place with the utmost rapidity. Contractions appeared synchronously in various portions of the calf muscles, and continued with such energy and rapidity that they suggested the movements of a mass of very lively snakes. Next to the calf-muscles, the muscles of the forearm and of the hand showed the most active movements. They were, however, much less striking in this region, for while some portion of the affected muscles exhibited fibrillary contractions almost constantly, the individual contractions were much more distinctly appreciable as such than they were in the calves, because only one or two portions of the muscles showed contraction at one time, while in the calves there was a maze of motion. The movements caused frequent twitching of the fingers, somewhat like a mild subsultus tendinum. They produced no movements in the feet or toes. The fibrillary twitchings elsewhere were decidedly less striking and less constant, and the individual contractions were so far separated as to time and space as to be individually quite distinct.

Besides this myokymia, the patient presented very slight persistent carpopedal spasm, most marked in the hands, which were always held in a position somewhat resembling slight tetany; the fingers were slightly flexed at the metacarpophalangeal joint, the thumb being adducted and in apposition. The position was not that of perfectly typical tetany, as the fingers were spread apart somewhat, the first finger in particular being decidedly abducted and occasionally strongly extended. There was a constant feeling of slight stiffness or contracture in the hands, and, except during the more violent spasms, the man was continually rubbing his hands gently in the attempt to relieve this stiffness. The anterior and posterior muscles of the calf, also, were constantly in mild spasm, the foot being held slightly rigid at right angles to the leg. The abdominal muscles, likewise, were practically all the time in very slight contracture.

The mechanical irritability of the muscles was greatly exaggerated. Very gentle palpation caused no spasm and was not painful. Moderately active palpation of the muscles; sudden, moderate pressure, or, even more markedly, a slight tap upon them would bring on violent and exceedingly painful local tonic spasm, during which the muscles would become board-like in hardness. At this time even gentle palpation of the muscles was painful. The spasm would last for fifteen or twenty seconds, and sometimes a little longer, and would then gradually relax, there being a distinct after-contracture, which took about half a minute to disappear.

Even a slight attempt at voluntary use of the muscles of the trunk or the extremities would often bring on violent spasm; and on attempting to sit up in bed the muscles of the abdomen became as hard and firm as a plaster cast, the spasm being exceedingly painful. Any active movement of the hands or the feet would bring on a spasm. I could not determine that there was any entirely spontaneous spasm. At times this seemed to occur, but usually, I think always, it could be referred to an attempt at voluntary movement. Mechanical irritation of the muscles, as by a blow, sometimes caused general spasm of the muscles of the extremities and abdomen; but this, I think, was due to the fact that the pain of the local spasm produced by the blow made the patient unconsciously throw his muscles almost immediately into action elsewhere, thus secondarily bringing on the spasm of these other parts. There was no spasm of the back or neck and no trismus. There was no abnormal mechanical irritability of the nerves; Trousseau's and Chvostek's phenomena were absent, as was Hoffmann's phenomenon.

There was no tenderness over the nerves. Changes in skin sensation were absent, and, except during the painful spasm, there were no subjective disturbances of sensation beyond some numbness of the hands. The knee-jerks and arm-jerks were slightly excited. The plantar reflex was not excited, and the epigastric and cremasteric reflexes were normal. Babinsky's phenomenon was absent.

I had no galvanic battery at hand, but excitability to the faradic current was greatly increased. The weakest current obtainable threw the muscles into violent spasm, just as did mechanical irritation. Tetanus was produced whether both electrodes were applied over the muscles, avoiding the nerves, or one electrode was put over the muscle and the other on the sternum.

The patient's mental condition was entirely normal, except that he displayed the slight soporosity to be expected with his fever. When I saw him his temperature was 101°; upon admission it had been 99.3°. Two or three hours after I saw him his temperature fell, and by the next morning it was normal, rising again that afternoon to 99.4°. Soon afterward it fell to normal and remained so subsequently.

Large doses of bromide were given, and also magnesium sulphate every hour until he was purged. The painful spasms disappeared entirely the evening of admission, and the fibrillary twitching became very much reduced. On the following day the patient was entirely free from painful spasm, and the muscles had quite lost their abnormal irritability to voluntary movement or direct mechanical stimulation. There was still fibrillary twitching in the calves and forearms, but of only slight degree. The urine, examined meanwhile, showed normal conditions, except for a

slight trace of albumin. The blood examination soon after admission showed: hæmoglobin, 93 per cent.; red cells, 5,640,000; leukocytes, 12,200.

The man remained entirely comfortable after this, and on the 6th demanded his discharge. At this time it was noted that his general condition was entirely normal. The tendon reflexes were normal. There was very slight fibrillary twitching in the calves and forearms, but the patient stated that this was usually present. The reactions to the faradic current were the same as were those in a normal man tested at the same time, and there was no abnormal irritability of the muscles.

CASE II.—L. S., colored, of unknown age (about fifty-five years), was admitted on July 1, 1903. I did not see this patient during his active symptoms, but Dr. Despard, at that time my resident, made careful observations and afterward, when the other patient was admitted, stated that the conditions were almost exactly alike in the two cases.

This man gave a negative family history and could not remember any previous disease. He had never had muscular cramps or twitchings. He said that this illness had begun after he had been hard at work and had become much overheated. He was seized with violent cramps in the muscles, first of the hands and then of the arms and legs. The cramps rapidly extended to all the muscles of the body, with the exception of those of the head and face. The attack came on at about eleven o'clock in the morning and he was admitted to the hospital late that afternoon. Meanwhile he had felt nauseated and had finally vomited once, and this had entirely relieved his nausea.

When he came in he exhibited a condition that need not be detailed, as it was practically the counterpart of that in the case already described. Painful spasms that were of brief duration but that occurred frequently affected most violently the muscles of the forearms and hands, and of the calves and feet; but also involved the thighs, the upper arms, and the trunk. Between the spasms there was constant and violent fibrillary twitching of the muscles of the extremities and of the trunk, that in the calf muscles being particularly pronounced and of much the same grade as in the other case. The mechanical irritability of the muscles showed the same excitation as in the other case. The electric excitability, unfortunately, was not tested at this time. Subsequently (when the patient was entirely over his spasms, however), the response to faradism was tested and found to be normal. As in the other case, the spasms were exceedingly painful. The patient showed no other abnormalities, except that there was a very high grade of arteriosclerosis, all the palpable arteries being hard, rough, and tortuous, and the urine showed the conditions to be expected with this —*i. e.*, a trace of albumin and a few hyaline and pale granular casts.

This man was purged, and nothing else was done for him. After having had a free bowel movement he was much improved, and by the following day was practically well—except that he still had a slight tendency to spasms and a very little fibrillary twitching in his muscles. Both the spasm and the twitching disappeared within about twenty-four hours after this. He was not tested during the attack for Chvostek's, Trousseau's, or Hoffmann's phenomena. The day after his admission, when I saw him first, and when he still had slight cramp, the phenomena mentioned were all absent. At that time the condition was so slight that I did not test the response of his nerves and muscles to electricity, doing this only later, when the admission of the other patient and his violent symptoms had made this man's condition of greater interest. The response to faradism was then, as stated, normal.

The cause of the phenomena in these cases is, of course, largely a matter of speculation. The short duration of the symptoms and the rapidity with which they disappeared after practically no treatment but mild purgation suggest a toxic origin. This statement, one that is perhaps somewhat too freely used nowadays when we are in the presence of the unknown, means in this case nothing more definite than that probably the high atmospheric temperature itself, or some little-marked digestive derangement produced by the heat, disturbed the body chemistry in some obscure way; although, on the other hand, I do mean by this suggestion that it seems improbable to me that the heat produced the condition directly, through mere irritation. The general body temperature of the first patient was but slightly elevated, except during a brief period; while the second showed practically no fever: and there are no observations indicating that elevation of the body temperature without toxæmia, even when the fever is very decided, tends of itself to excite spasm. The more or less pronounced spasm not infrequently seen in acute infections is almost certainly due less to the fever than to the toxæmia. There is, then, no good reason to believe that the slight elevation of body temperature exhibited by these patients had any direct relation to the spasm.

It is of couse possible that the immediate effect of the external heat upon the skin may have excited the spasm reflexly, but this seems to me an unsatisfactory explanation; for greater heat than this when applied in the form of hot baths tends to relax such spasm rather than to increase it; and in these patients hot baths had never produced any tendency to spasm. I have, however, already referred to the fact that in the cases of Kny and Schultze, and in two of Talma's exposure to cold or to cold and wet excited the spasm, and I have also noted the effects of cold water in my first case. These observations, and particularly the common con-

ditions in swimmers' cramp, suggest that reflex irritation alone might possibly have excited the spasm.

This question is one that cannot be definitely settled. It is somewhat analogous to the nature of tetany, in connection with which there has been so active a discussion as to the relation of reflex action and toxæmia in the production of the spasm. In tetany the most satisfactory evidence certainly points toward toxæmia; and I think that the same is true of most of the acute and subacute cases of other forms of myospasm.

It is conceivable that the heat itself had nothing directly to do with the myospasm, that both my patients had some digestive disturbance that was but little evidenced by symptoms, and that toxic material absorbed from the digestive tract set up the spasm; but in the absence of anything more than nausea and slight vomiting, especially as these symptoms came on only after severe spasms had lasted several hours, such a source of the toxæmia, while possible, seems to me improbable and at best purely suppositious.

It at first occurred to me that, as they probably are in cholera, for example, the spasms in these cases might have been due to loss of fluid. If, however, this cause was active in my cases, it must have been the result of excessive sweating, as diarrhœa and severe vomiting had not occurred. Since I could not determine that either patient had been sweating excessively, this cause seems very improbable; and the blood count in the first case, although slightly high, is distinctly against such a view.

There is one other factor of sufficient interest to deserve mention, and this is the high degree of arteriosclerosis present in the second case. Walton has laid special stress upon the relation of arteriosclerosis to myospasms, and it is not improbable that in this case it was an important predisposing factor. In the first case there was apparently habitual predisposition; there was certainly no evident arteriosclerosis.

As to the classification of these cases: Any attempt to carry this out definitely is, I think, a somewhat fruitless task, except that it may be stated that they belong to a heterogeneous group, and will not fit well in any place in a schematic classification. The patients showed severe tonic intention spasm and presented at least several features of the myotonic reaction. Following Talma, therefore, and dwelling especially upon the tonic spasm, they might be classed as transitory acquired myotonia. On the other hand, Kny classes his cases as most closely related to paramyoclonus multiplex of Friedreich; and Dana agrees that myokymia is best placed in this class. Talma's name for his cases, however, is hardly justified by any intimate relation to Thomsen's disease, and Dana's view is strongly opposed to that of Gowers and Walton, and it seems to me that the latter authors justly exclude myokymia from the class that includes paramyoclonus multiplex. That my

cases have any serious connection with either Thomsen's disease or paramyoclonus multiplex seems to me to be almost out of the question.

If one is to make a definite classification merely on the basis of certain striking clinical symptoms, these cases might equally well or better be brought into a group with tetany. In a number of points they clearly resembled tetany; but, on the other hand, the tonic spasms were almost entirely intention spasms; the myokymia was quite as marked a feature as the tonic spasm; considering their severity, the attacks were remarkably brief for tetany; and much more important than these facts, the Chvostek, Trousseau, and Hoffmann phenomena were absent. Hence, these cases certainly cannot well be classed with tetany.

The variety of classifications adopted by various authors when dealing with the myospasms shows that the manner in which many of these conditions are grouped is largely a question of personal fancy. This can, it seems to me, scarcely fail to continue to be the case until the nature and the manner of causation of the various myospasms become clearer than they are at present.

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A CASE OF PAPILLOMA OF THE BLADDER, COMPLICATED WITH PYONEPHROSIS, WITH REMARKS ON THE SURGICAL TREATMENT OF PAPILLOMA.[1]

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G. W., aged fifty-seven years, had noticed that for several months his urine had been mixed with blood; but other than a certain amount of weakness following these hemorrhages and a dull pain in the left lumbar region, the patient feels fairly well. Upon questioning, however, he complains of certain vague dyspeptic symptoms and dryness of the mouth. No headache. Sleep is uninterrupted, and examination of the thoracic viscera reveals

[1] Read by title at the meeting of the Urological Association of France, Paris, October, 1904.

nothing abnormal other than a slight chronic bronchitis. Pulse 80, hard, and rather tense. Some general arteriosclerosis. The urine contained a considerable amount of pus, blood, and renal cells from the pelvis of the kidney.

Cystoscopic examination of the bladder showed a body situated on the left side of the bladder wall in the vicinity of the ureteral orifice, but its exact nature could not be made out on account of the clouded condition caused by the presence of blood.

After several days of preparatory treatment of milk diet and the exhibition of urotropin a transversal suprapubic incision was made, and the bladder opened, and by digital exploration a polypus of soft consistency and distinctly pedunculated was found seated in the floor of the bladder to the left of the middle line. In size it might have been that of a large walnut.

The growth was seized as near its base as possible, and its pedicle was excised along with a certain amount of the surrounding vesical mucosa. Sutures were then inserted, and the resulting wound closed without much loss of blood.

On account of the presence of pus in the urine it was decided to drain the bladder. The suprapubic wound was loosely packed with iodoform gauze, and a permanent catheter was inserted.

The after-course of events was simple. The catheter was removed at the end of the sixth day, and the iodoform gauze packing was changed daily until the eighteenth day, when only a small opening remained through which very little urine was voided, the greater proportion being expelled by the urethra. In four weeks' time the suprapubic opening had completely closed, and the patient was discharged.

The patient was seen three months after the operation, complaining of pain in the left lumbar region, recurrence of the hæmaturia, and chills. The temperature was 38.8° C., and the pulse was weak and irregular at 110. The suprapubic incision was found in excellent condition, firm union having taken place.

The urine contains a large amount of blood and pus. Tongue dry, and digestive disturbances, which had somewhat improved before the patient left the hospital, had returned with greater severity than before.

By palpation an enlarged left kidney could be easily detected, and it was thought that fluctuation could be elicited.

The next day the kidney was exposed by a transverse incision and freely incised, which gave exit to about 500 c.c. of dark-red and very ill-smelling pus. Two large drainage tubes were inserted and the wound packed with iodoform gauze.

Within a day or two the temperature had dropped to normal and the pulse to 90, and was of better quality. The patient also commenced to recover his strength, and was able to take a fair amount of food. From this time on the progress toward recovery

went on uninterruptedly, and he was discharged, with a renal fistula, five weeks later.

Eighteen months after the last operation we saw the patient in excellent health, but still discharging a certain amount of urine, and some pus by the fistula from the kidney. But on account of his excellent condition and the absence of all symptoms pointing to uræmic poisoning, we have thought it best to desist from doing a secondary nephrectomy, which can always be resorted to should the indications point that way.

In considering the history of this case it would appear to us that in all probability there was a relationship between the papilloma and the suppurative renal process, inasmuch as the vesical neoplasm probably obstructed the ureter, causing a retention, and the kidney thus weakened easily became a prey to secondary infection.

Microscopic examination of the neoplasm showed the ordinary picture of papilloma without any evidence of malignant transformation.

I would like now to discuss briefly the surgical treatment of papilloma of the bladder in a general way, and, although I have nothing novel to offer, I can at least describe those methods which at my hands have proven the most satisfactory.

Small pedunculated papilloma in the female may occasionally be successfully removed without much difficulty through the dilated urethra; but this manner of operating I believe to be unsurgical, for the simple reason that one is working in the dark, and if the urethra is dilated to the extent requisite for giving a large enough operative field, permanent incontinence of urine is the usual inevitable result, and for this reason I believe that the suprapubic incision is the proper one to select, as I have already pointed out in a paper entitled "Chirurgische Behandlung des erworbenen unwillkürlichen Harnabganges bei Frauen," and published in the *Monatsschrift für Geburtshülfe und Gynäkologie*, Band xix., Heft 4.

Other intravesical operative procedures applied to the male have been described from time to time, such as Chismore's aspiration with a metallic catheter, etc., but all such methods are defective, inasmuch as the operation is performed entirely in the dark. On the other hand, Nitze's cystoscopic technique is certainly a vast improvement over the older manœuvres, and, although this distinguished *confrère* has published thirty-one cases in which he obtained excellent results, it would appear that this ingenious invention has not found many supporters.

The reasons why Nitze's method has not been adopted in general is because the removal of a large papilloma requires a number of sittings, and when one is dealing with a patient already in a weakened condition, the *séances* can only be undertaken at considerable intervals. For most surgeons this is enough to deter them from undertaking the removal of a papilloma by this method, and, on

the other hand, I believe that the majority of patients would prefer to take a little ether and have the neoplasm removed at once and for all than to be continually tampered with.

I would also point out that Nitze himself admits that accidents may occur during or after the execution of these intravesical operations, such as severe hemorrhage or cystitis, irritation of the kidney or its pelvis, as well as febrile disturbances, which simply mean a septic condition; and, last of all, but by no means the least important, prostatitis or epididymitis has been known to supervene. On another occasion Nitze says that severe hemorrhage from the growth, severe cystitis, a small bladder, etc., will render the intravesical operation impossible, and if the neoplasm is of large size, or when there are several growths, the execution of the operation is extremely difficult.

Now, if such an expert as Nitze himself is in these matters admits such difficulties in the operation, it is quite natural that the average operating surgeon will select another technique on which he can rely with practically perfect security. It is evident that many of the present defects in Nitze's method may be removed by the improvement in the instruments, so that the future of this procedure may be increased, but for the present it would appear that its application is extremely limited.

In opposition to intravesical operations we have three techniques by which the tumor may be exposed and removed. The first and oldest of these is Thompson's median perineal incision, which at the present time has lost its diagnostic value on account of the general use of the cystoscope, while its therapeutic importance for the removal of papilloma is practically *nil*, but its applicability for the removal of calculi may be still reserved for certain cases. This procedure may give satisfactory results to a few picked operators, but it is to be doubted whether it will ever be generally employed, because, on the one hand, there is always considerable danger of accidentally wounding the bladder, and, secondly, one never can be sure that he has completely removed the growth. The demands of modern surgery that the field of operation should be freely exposed will certainly place this method in the background, more especially so when one recollects the great possibility of hemorrhage during the operation and the very great difficulty one would meet with in controlling it through the perineal incision.

Koch collected thirty cases operated on by Thompson's method, and in only two was the complet eremoval of the neoplasm a certainty. In five no recurrence had taken place after the first year, while in seven no symptoms returned for two and one-half years; five patients died during the first few weeks following the operation, death being partially due to exhaustion resulting from the severe hemorrhage during the operation, and partly from some intercurrent affection. In more than six of the cases no opinion

could be formulated on account of the short lapse of time since the operation, while in three cases a recurrence took place for the simple reason that the neoplasm was only partially removed. In one case there were three recurrences due to an incomplete removal of the growth. It would seem from these cases that an entire extirpation of the neoplasm is not possible to attain excepting in a few instances unless the tumor grows near the urethral opening of the bladder or in the pars prostatica, under which circumstances König is of the opinion that the perineal incision is more advantageous than the suprapubic opening.

Colpocystotomy for the removal of papilloma in the female bladder has only a limited field of usefulness. The operation is impossible when the vagina is small and also when the tumor grows from the anterior wall of the bladder. Then, again, large-sized growths can only be removed by this route with difficulty, and then only by resorting to morcellation.

Suprapubic cystotomy is in all probability the method now generally employed for the removal of papillomata or other vesical growths. Combined with Trendelenburg's position it fulfils all the requirements for the complete exposure of vesical neoplasms, because a complete view of the entire cavity of the bladder may be obtained, and all parts are accessible to operative manipulations. There is no doubt in my mind that by this route the greatest convenience and security are to be obtained, and as far as hemorrhage is concerned, there is no other technique which gives such absolute security as this one.

The defective points in the operation are, first, the danger of opening the peritoneum, and, secondly, urinary infiltration of the pelvic cellular tissue; but the progress of surgery has practically done away with the first danger, so that should the peritoneum be opened it can be immediately closed, and no ill result is apt to accrue. The dangers of infiltration of urine can be, I believe, easily avoided by proper drainage.

It would also seem to me, from a very large experience, that the transverse suprapubic incision is far more preferable than the median one, and in corpulent patients it certainly gives a far more extensive field of operation.

To render the bladder more ready of access, the injection of air is, to my mind, better than sterile liquid, because, when the latter runs out through the abdominal wound, it inundates the entire field of operation, and if the bladder is infected, the liquid carries the agents of infection with it, which may infect the cellular tissue. Petersen's rectal bag should never be employed, being both useless and dangerous.

The removal of the papilloma, whether pedunculated or not, should include the removal of a certain amount of mucous membrane around the base of the growth, followed by a careful suturing

of the remaining defect. Some surgeons are satisfied by simply ligating the pedicle, cutting off the growth, and then applying the thermocautery to the stump; but this procedure is not safe, because one can never tell whether the growth has commenced to undergo malignant transformation, and in its removal it is more prudent to assume the possibility of malignancy.

Regarding the dangers of purulent infection of the prevesical cellular tissue, I believe with Kraske, that the use of the permanent catheter is not alone sufficient, and that unless the bladder is free from cystitis it is all-important to obtain free drainage. If, however, the urine is apparently aseptic the bladder and the abdominal wound should be closed by a careful suturing, and the bladder drained by the use of the permanent catheter.

To effect a perfect drainage of the wound, the use of iodoform gauze loosely packed in and changed daily is, to my mind, the proper way, and secondary union will follow quickly. The resulting cicatrix is firm, and urinary infiltration is avoided. This manner of drainage is to be recommended in cases of purulent cystitis and, in fact, after all operations where bladder drainage is required for a short time following an operation. Combined with this, the use of a permanent catheter should be resorted to as long as the patient can comfortably bear it and its presence in the urethra gives rise to no untoward symptoms.

SOME UNSETTLED AND IMPORTANT PROBLEMS IN THE TREATMENT OF ACUTE LOBAR PNEUMONIA.[1]

By Beverley Robinson, M.D.,
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To the practitioner of medicine there is no subject of greater interest than the treatment of pneumonia. This interest proceeds from the facts: 1. That pneumonia is met with frequently in all climates and seasons, everywhere, in hospitals, and in private practice. 2. It is a very fatal disease, of rapid course, and of markedly infectious type at times. 3. It has been the subject of a great number of books, papers, discussions from the ablest and most experienced physicians in the past and at the present time. 4. While there is a consensus of good medical judgment as to the efficient cause of pneumonia, as to many accidental and co-operating causes, as to certain indications of treatment, and as to usual causes of death due to it, there still remains much that is undetermined in its treatment, and about which many "good men and true" hold divers opinions.

[1] Read before the American Climatological Association, June 4, 1904.

In my remarks I wish to be understood as limiting the discussion as far as possible to acute lobar or croupous pneumonia, with a consideration solely of the usual dangers or complications occurring in its course.

I would merely add, for additional clearness in the beginning, that certain cases of pseudolobar pneumonia are with difficulty differentiated from the true form, either by symptoms or by physical examination. It is true that the debut of the pseudo form, the temperature chart, the previous bronchitis, the absence of herpes and flushing of the face, the bacteriological findings of several kinds of micro-organisms, and not merely the pneumococcus of Fraenkel, and the isolated areas of consolidation preceding, it may be, and probably recognized before the lobar consolidation is formed, will permit, as a rule, an accurate differentiation to be made. And yet there are cases, as I believe, in which the most careful clinical observer may remain somewhat undetermined as to which form of disease he has to do with for one or more days. Indeed, the autopsy alone has in rare instances definitely solved reasonable doubts.

In this discussion I do not propose to deal with statistics of my own or even of others which I might readily obtain. This has been done already many times, and in the aggregate in a vast number of cases. I regret to say that the resultant figures have not been very encouraging, nor have they shown great progress in treatment; nor have we seen, apparently, the virulency or the mortality of the disease decrease, if we carefully consider the records of a certain period of years in the same or different places. Besides, it must be admitted that statistics as made unfortunately do not tell, never will tell, perhaps, precisely what should be a true and proper estimate of prognosis in any given case of pneumonia. Personality, circumstances, antecedents, temperament, constitutional peculiarities or defects, all differ or are of untold variation. Again, as we all know, epidemics of pneumonia, or the endemic, sporadic sort, show more or less virulence or intensity of poison, depending upon causes, of which we know now, as heretofore, very little in an accurately scientific manner.

Before I speak of problems in curative treatment, let me say a few words in regard to prevention of pneumonia. I do not wish to dwell on facts about which we are all pretty nearly agreed, namely, to avoid excesses, overfatigue, chill, bad air, etc., but upon some which later investigations of others and personal observations render valuable.

We know to-day that the pneumococcus is found in the secretions of the throat and mouth of many healthy people and for long periods of time. One or more of the contributive causal conditions referred to may render these micro-organisms virulent or infective. Is it not specially indicated, therefore, to make use of antiseptic gargles

and mouth-washes frequently when we have reason to fear the possible beginning of pneumonia? This antiseptic mouth-wash, at such times, should have a slightly acid reaction, simply because we know that the pneumococcus does not thrive in an acid medium. Incidentally I should add that, on account of the deleterious action upon the teeth, I do not approve of a mouth-wash of acid reaction to be used habitually in a condition of normal health.

With the first symptoms of chill, fever, pain in the side, cough, and expectoration, I believe beechwood creosote should be vaporized more or less continually in the patient's room. I am confident there is no drug used in this way which has more preventive value in the treatment of pneumonia. Of course, I cannot prove to others that several times already by this means I have prevented in private practice, as I believe, what otherwise would have become pneumonia. Whether it would have become croupous pneumonia or bronchopneumonia, I am not prepared to say except in a hesitating way, and with, it may be, a conviction tending toward the latter opinion. I wish it to be understood that I only give to creosote a very slight or moderate antiseptic power locally; but I know it is an anticatarrhal agent of great value directly and by elimination through the bronchial mucous membrane. Further, I am thoroughly convinced it is the best way to give creosote so as to obtain its constitutional effects without disturbance of the stomach, and, as a rule, without injury to the kidneys. It is wise, of course, if creosote be used freely in inhalations, to examine the urine carefully and frequently for any appreciable amount of albumin. If this be present we should try to determine its cause—whether it be the result of the pneumonia or of the action of the creosote, and act accordingly.

Occasionally, creosote by inhalation used too freely gives headache to the patient or to the nurse. In this case it should be intermitted for a few hours, perhaps, and the room thoroughly ventilated. It is also my belief that creosote thus used prevents the taking of pneumonia by the nurses in attendance or by any near relative very constantly in the room.

The degree of risk of contagion in *the air* is, of course, a questionable matter, and for my part in the majority of instances I doubt the reality of it. In other exceptional instances and in certain epidemics when the pneumococci are more abundant or more virulent, or both, than usual, the transport of the noxa of the disease through the air no doubt occurs, and may be best neutralized primarily by insisting upon having the vapors of creosote in the room. Of course, the sputa should be rigidly and systematically disinfected, and the lips and other parts of the face which have been soiled with them properly cleansed. So, also, must all soiled garments and bedclothes be suitably disinfected upon removal, and after the recovery or death of the patient the furniture and room should be properly

fumigated, cleaned and ventilated, just as we should do in the case of any other possibly virulent contagious and infectious disease. The sputa should never be allowed to become dry, but should be received in a convenient receptacle containing a solution of carbolic acid or bichloride of mercury. If the pneumonia becomes developed I believe antiseptic mouth-washes and creosote inhalations should be continued throughout the acute stage of the disease, and, indeed, until the local consolidations have been resorbed.

The necessity of a large well-ventilated room, with open fireplace if possible, of two well-trained, careful, judicious, and attentive nurses, of proper assimilable food (mainly milk), of a sufficient quantity of good drinking water, and of due attention to the emunctories, namely, the skin, bowels, and kidneys, is all-important, but need scarcely be insisted upon with my audience.

Now we come to some points of judicious curative treatment of pneumonia which are very important, and about which also there may be very different judgments. Among the poor it is difficult, almost impossible, to treat pneumonia as it should be treated, because of the lack of proper surroundings and nursing, not to speak of food, drugs, and very good medical care. Under these circumstances is it proper to transport the patients to the hospital? In general, if the patient be seen at the beginning of the attack, I believe we should do so. Later, at the third, fourth, or fifth day of the disease, to my mind, it is often questionable. Once in the hospital bed such a patient will undoubtedly get better care than at home, and on this account has a better chance of recovery.

On the other hand, when we consider the fatigue, the exposure, the unavoidable nervous upset incident to this change, it becomes a nice question to decide at times, and there is no absolute rule to lay down in advance. The patient and the doctor must decide. If the patient still be reasonable and not too critically ill he may go to the hospital if he desires, and the doctor may consent, if the hospital is not too far removed, an ambulance be had, proper stimulants be given before removal, the patient be thoroughly covered and the removal be conducted in a quiet, expeditious, and thoroughly efficient manner. If the room and surroundings be tolerable and the care of the patient be fair, although not of the best, proper duty to the patient must be decided upon and carried out, after careful and due consideration of everything involved in the individual instance.

When the patient reaches the hospital should he be treated in the general ward? Not so, if it can be otherwise ordered. His chances of recovery are not so good as in a private room or in a small ward where there are only two or three other patients. The noise of the ward is objectionable; the light of the ward is also objectionable. Noise is unavoidable here; light can only be subdued by cutting off air in a measure; inhalations cannot be carried out as

well and as efficiently. I have tried and know of what I speak. So far as other patients are concerned, *pneumonia patients* should be isolated; not because all patients are infective or contagious to others, with proper care and precautions, but a few possibly or probably are, and in time of a prevailing and increasing epidemic influence this should be considered, and more than usual protective care to others should be insisted upon. Of course, I do not wish arbitrarily to ignore the difficulty of doing this, especially if there be numerous pneumonia patients, as has been true during the past winter and even now. Nevertheless, the indications should not be lost sight of. On the contrary, they should be insisted upon, and as far as possible carried out.

A word, now, in regard to the results of treatment in hospitals. Many times it is most unsatisfactory. Again, in certain seasons, we get a series of cases which do well almost invariably, and really with very little active treatment of any kind. In fact, a wise expectancy seems to be most judicious and to afford very good, perhaps the best, results. Why, indeed, should we expect good results from hospital treatment as a rule and when the cases of pneumonia are of a virulent type? They are brought into the hospital usually at a time when the disease is well advanced (second, third, fourth, or fifth day), sometimes almost moribund, and we begin to give our stimulants or other remedies. Already the patient has had, in addition to an acute and virulent disease, imperfectly and perhaps very ignorantly cared for at home for forty-eight hours or more, the *risk* of having gone through all the anxiety, fatigue, exposure, etc., of the transportation to the hospital. Further, we have often to do with poorly nourished and broken-down subjects prior to the attack —subjects, it may be, already of disease of heart or kidneys. Too frequently they are victims of alcoholism or some drug habit. Again, they are of all ages, and the very young and those of advanced years are of the number. Given all these conditions, how may we fairly compare results of treatment thus obtained with what we should obtain, with what we do obtain sometimes, many times, it may be, in private practice?

There is one other thing to which I wish to direct careful attention in this connection, and one far too often overlooked practically in the treatment or estimate of every virulent disease, and particularly when the poison is very intense in quality if not in quantity. *The time of the treatment* when begun is most important—essential and decisive at times in regard to the ultimate outcome of the disease. By doing the right thing at the right time life may be saved; when there is delay in doing the right thing life may be lost. The analogy here which I would refer to is the use of antitoxin in toxic diphtheria. Made use of in the beginning in sufficient doses, the patient will probably recover; delayed in its use, or given in too small amount and not repeated, the patient may die. The patient

will *surely die*, sometimes even with the help of a most wonderful remedy. So I believe it to be in a measure with the treatment of acute lobar pneumonia. Put your patient to bed immediately on the advent of serious symptoms of the disease in a large, sunny, well-ventilated room, provide him immediately with the best nurses and good milk and water; begin immediately antiseptic inhalations, and use all our other curative means sensibly, judiciously, appropriately, and I believe many bad cases may be saved. But delay these primary and efficient, though simple, means too long and the patient will often die. He will die because the poison of the disease has taken hold of him too strongly, in too large quantity, and before proper and suitable means have been employed to check or throttle it.

Now, there are some cases of pneumonia, as there are some cases of diphtheria, enteric fever, scarlatina, smallpox, etc., which will almost inevitably die to-day, as always up to the present time, no matter what treatment is employed. And the proof of it lies in the record of medicine for one hundred years or more. Indeed, the virulency of some of these cases is such as to render them well-nigh hopeless, and they will remain so unless medical science gives us curative sera superior to any we now possess. We must also bear in mind here and ever that we shall have occasionally to do with what occurs both in hospital and private practice, namely, the complications of the primary disease, such as Bright's disease, pericarditis, meningitis. These diseases being present, the case must be managed rationally and in view of everything involved. Despite the best care and treatment, they add much to the gravity of the prognosis, and in some instances they make recovery almost impossible.

One of the most important problems connected with the treatment of pneumonia is the safe and proper use of opium or its alkaloid, morphine. In the beginning, when the pain in the side is intense owing to the complicating pleurisy, it is occasionally indicated to give a hypodermic injection of morphine to allay or arrest suffering. I find little or no objection to the use of the drug in moderate dose at this time, provided the kidneys have not been diseased previously. When the pain is only moderate I prefer local applications as being frequently sufficient to diminish or stop the pain, and because they are useful doubtless in limiting local congestion of the lungs. Later in the disease (and up to the second, third, or fourth day), and despite the judgment of many good observers, notably Dr. Samuel Wilks, of Guy's Hospital, who feels happy only when five grains of Dover's powder are given every four hours in pneumonia, I prefer to abstain from its use except in the rarest instances. I do not believe the opiate has any good effect upon the march of the pneumonic process; I do believe it is prone to lock up secretions which are already too much interfered with by the disease itself. Professor William

T. Gairdner notably has directed attention in the grave and advance forms of pneumonia to the great and immediate danger resulting from the use of an opiate. To dull the respiratory nervous centre at such a time, when nature is striving all she can to protect herself by increased frequency of breathing against inroads of a disease which prevents proper oxygenation of the blood, is to hasten the death of the patient or at least to diminish his chances of recovery. Dr. S. S. Burt has again directed attention to this subject, and in my judgment very wisely. Of course, distinctions should be made. If the patient be of middle life, with previous evidences of interstitial nephritis upon which a marked acute process is grafted, we *must* do without an opiate.

In more than one instance, I fear, I have hastened death through ignorance in permitting its use. When the kidneys are acting well, with little or no albumin, casts, or blood present, if delirium or insomnia are pronounced and threatening, and if other means have failed to relieve, a small or moderate amount of morphine hypodermically is permissible.

Personally, I have never believed in the cold bath, cold pack, or even cold applications locally in the treatment of pneumonia. Occasionally I might permit cold sponging if the fever were very high, but, as a rule, I much prefer the use of tepid or warm water for bathing purposes, to which a moderate proportion of alcohol is added. It seems to me that efforts to diminish fever *per se* in the treatment of pneumonia are scarcely ever a primary and essential object, because I do not believe, as a rule, that the muscular changes produced by increased temperature in its relatively short duration, although very high at times, can be very pronounced; and I believe the nervous shock caused by the toxin is better allayed with slight or moderate heat than with cold.

For a somewhat analogous reason I am opposed to all large doses of quinine to abate pneumonia or to act as a pronounced antipyretic during the course of pneumonia. On the other hand, I thoroughly believe in moderate doses of quinine when well tolerated by the stomach, as a tonic, as a slight antipyretic, as a blood disinfectant, throughout the course of pneumonia. In large doses, while quinine does diminish temperature, it also decreases heart power and general strength, and, therefore, is prejudicial to the patient's well-being. Be it added, however, that quinine as an antipyretic for those who wish to follow out this indication formally is far less dangerous in use than antifebrin, antipyrin, or even phenacetin.

I am of the opinion that there is weighty evidence to prove that the solution of ammonia is a preparation, used moderately and continuously in pneumonia, of considerable value. Its value is specially shown not as a stimulant—because in that particular I believe alcohol, as whiskey or brandy, to be more desirable—but to keep the blood alkaline and fluid. In this disease, especially in the grave

forms and where there is extensive pulmonary consolidation, it is to my mind a very important matter. I have seen many patients in whom, before death and at the autopsy, I have been convinced that heart clot avoided would possibly or probably have meant a life saved. To no one as much as to Benjamin Ward Richardson is the honor due of great insistence upon this important fact, too much ignored to-day in medical writings and medical practice.

Large doses of digitalis at any period of the disease are, as I believe prejudicial to the patient and can never do good. In the milder forms of the disease they are not even indicated from any point of view; in the graver forms and in middle adult life, particularly of most city men, arterial changes counterindicate its use, not to speak of possibly granular degeneration of cardiac muscular fibre. Frequently I have seen the pulse become rapidly very tense and more or less irregular under its use, and the heart action tumultuous, irregular, and forced, as it were, uselessly and vainly. Minute continuous doses of digitalis, given before there are any symptoms of heart failure may prove useful in warding it off, and, when heart failure shows itself not discontinued but protected as to its action upon peripheral arteries, with a moderate amount of nitroglycerin, $\frac{1}{200}$ or $\frac{1}{100}$ grain given every two or three hours, I have found useful, I am confident.

We must bear in mind always that digitalis is not the drug from which we should expect good effects very rapidly in the treatment of pneumonia. Its action, to be useful, must have time as an element, and if given in small doses its cumulative effects, by reason of slow elimination, do not become prejudicial. Apart from other considerations, digitalis in anything but small doses will often cause stomachal intolerance. So soon as there is evidence of pronounced cardiac weakness, digitalis, or, much better, good digitalin and strychnine, should be given hypodermically, and not by the mouth. Medication by the mouth in grave conditions in pneumonia is uncertain and unreliable, except for alcohol and possibly nitroglycerin, which do not derange the stomach, and hypodermically may cause considerable local irritation.

In regard to strychnine, while I value it highly in the treatment of pneumonia, I am satisfied we have to-day often pushed its use to an extreme degree, and in more than one instance in which a $\frac{1}{30}$ or even $\frac{1}{20}$ grain had been used hypodermically every two or three hours, I have cried a halt, because I was satisfied insomnia and delirium were being aggravated and the heart action not in reality stimulated by this overdose. I am inclined to believe that my friend Dr. R. G. Curtin has struck a warning note in this connection which the profession will do well to consider and be guided by. In more than one instance in the treatment of pneumonia, and where other heart stimulants had been of little or no avail, I am sure I have been of use and helped save life with the use of the best available prepa-

ration of coca. Given by the mouth or hypodermically, it has often been of service when I had almost given up hope. The reason, I believe, that this agent does not more frequently respond to our wishes is because the preparation employed is relatively inert. Many coca leaves, not to say the far greater majority, are dry and of poor quality when first gathered. Further, the wrong variety of leaf from which to obtain the tonic alkaloid of the plant is usually employed. Hence, much of the coca used, as wine or extract, contains a large percentage of cocaine, which I believe has little or no tonic properties, and does not contain the other derivatives most valuable in cardiac exhaustion of pneumonia. Here is not the time or place to speak further on this topic. It is wisdom to give black coffee frequently in advanced cases of pneumonia with evidence of heart weakness or cardiac degeneration, whenever our coca is unreliable, either by mouth or rectum.

There can be little doubt that saline solution infused by the veins or by hypodermoclysis or by the rectum, after a moderate bleeding, has been useful in some instances. I must give a warning note as to using too much saline solution by the veins or too rapidly. I have seen death thus occasioned, as Tyson has also, presumably by dilatation of the right heart.

There is no doubt in my mind that oxygen given frequently from the time pneumonia shows marked gravity is extremely beneficial in many instances; but to be of real value it must be given freely and with unstinted hand, and not begun when death is near and already the patient almost moribund. As to a good preparation of oxygen causing notable bronchial irritation, as is sometimes stated, this I have rarely seen, if at all.

There is a point to which I would refer in connection with treatment, because from its appreciation may proceed, I trust, something of real value in prevention of imminent danger. Absence of leukocytosis in acute lobar pneumonia presages death, as hitherto observed. It behooves us, therefore, to have a careful, differential blood count made as soon as the disease is at all clearly defined; and no matter how limited locally the disease may be for a while, or how little threatening at first the symptoms, we cannot be too solicitous or too careful in such cases in our care and treatment. Several times death has followed rapidly, in my experience, and except for the known result of the blood count I should have been lulled into a false security. Later, also, we may obtain facts of great value from the differential count of the leukocytes, as we now do in appendicitis. If in the beginning, for example, we discover the polynuclear neutrophiles in relative excess and the eosinophiles notably decreased or absent, may we conclude that these are, in reality, the very serious cases, "whether associated with an increase of the total number of the leukocytes beyond the normal or not?" I must leave a positive answer for the while with my laboratory friends.

I am of the opinion that except in the beginning of a pneumonia, and in specially rugged or full-blooded patients, venesection is not desirable, as a rule. To this there is one exception. In the first two or three days of the attack of acute lobar pneumonia where general œdema of the relatively healthy lung occurs, I have known life to be saved more than once by a prompt and sufficient venesection (eight to twelve ounces as a mean). Later in the disease, while at times it might be indicated and performed, the result would rarely prove beneficial for more than a very brief period. I grant only two exceptions at an advanced stage. One is, where the right heart is distended and unable to cope with its fluid contents, and despite all judicious cardiac stimulation, the use of nitroglycerin, oxygen, etc.

In these instances I have seen temporary good effects produced by a venesection, and occasionally, perhaps, a life saved. The distention of right heart and jugulars, with great breathlessness, pallor, or almost lividity of lips, face, extremities, and even portions of the trunk, means beginning thrombosis, and reducing the bulk of blood will not save life unless we dilute it, and incidentally, perhaps, partially neutralize its contained toxins. Give, therefore, a moderate saline infusion after the venesection, with this hope, and also insist upon beginning immediately or continuing frequently the use of ammonia under form of the strong solution or the aromatic spirits.

The other condition in which venesection may be and is occasionally useful is where the patient is suffering unquestionably and mainly from profound general toxæmia. It is then an indifferent matter, relatively, whether the pneumonia be in its initial stage or fully developed. In the latter stage of the disease, as in the former, I should usually consider it indicated to make a moderate infusion of saline solution by the veins, or give the solution by hypodermoclysis, or by repeated enemas or irrigations, to dilute the poisons remaining in the blood. I insist, however, that the quantity of the venous infusion in these and all instances should be moderate and the infusion slowly made; otherwise the benefit derived from the venesection will be rapidly neutralized and the patient's immediate condition rendered even worse than prior to the active interference. In one case that comes vividly to mind I felt satisfied that the excessive and too rapid infusion certainly hastened the fatal termination of the case. After the venesection the patient showed signs of returning consciousness, although just before in active delirium, and the pulse was decidedly stronger. A little later, after the venous saline infusion, unconsciousness returned, and the pulse lost strength perceptibly and rapidly until death supervened.

There are good reasons at present to support my contention, particularly in pneumonia. In this disease there is a notable retention of chlorides; hence we should not add much to these, especially where there is doubt as to the condition of the kidneys. If there be

marked epithelial changes in these organs, which as we know not infrequently exist in severe cases of lobar pneumonia, rapid œdema of the lungs may be effected, which shall prove to be the immediate cause of death, or of an œdema of the meninges or other serous membrane, which may cause a rapidly fatal termination of the case.

The quantity of water may do harm. Whenever saline infusion is followed by marked diuresis we are apt to obtain good effects; but in other instances we find the contrary prevails and certain accidents occur. Some of these, due to a further retention of chlorides, I have already referred to. But we may have others, occasioned especially by the amount of the fluid itself, which are dilatation of the right heart, followed by cardiac failure; vascular distention in the lungs and, as a result, great dyspnœa, accompanied or not by pulmonary œdema, and uræmic symptoms of imminently grave import, the explanation of which is probably multiple. Suffice it to add that saline infusions, moderate in quantity and slowly made, are cardinal indications in my judgment, at present, whenever this treatment is seemingly called for in the treatment of lobar pneumonia.

It may be that in certain instances it would be well even to substitute the sulphate infusion for the chloride, in view of the injurious known results of the latter at times, and although the quantity infused is moderate. This problem is still *sub judice*.

No doubt in the beginning of the attack of acute lobar pneumonia it is useful to prescribe a few grains of calomel to free the intestinal tract, thus lessening local congestion of the lungs and eliminating a certain amount of toxins. Aside from this it is claimed that a dose of calomel, if sufficiently large and employed properly, may act as a local disinfectant of mouth and throat (Musser), and later, in like manner, in stomach and bowels. At an advanced stage of the disease we all know what extreme gravity is connected with great distention of the bowels, whether it acts mechanically by pressure upon heart and lungs, and thus prevents their functions being exercised, or by reason of great absorption of toxins from the bowels, increasing notably this source of peril to the patient. In either event we have often a paralytic condition which aids further distention and is only too evident and threatening. When this condition occurs we should first of all empty the bowels by a proper dose of some mercurial, preferably calomel or a gray powder or a saline aperient, and afterward give a pint or two of normal hot saline solution as an injection, which may be repeated twice or more times in twenty-four hours. Not only is much decomposing and poisonous excreta thus carried off, but, in addition, through absorption, the toxins in the blood are diluted and rendered more bearable by the patient, and at the same time the heart action is strengthened and the renal secretion rendered more active.

By some we know the kidneys are considered the most powerful emunctory of the economy in acute lobar pneumonia, and whenever

the urine diminishes notably in amount and there is great decrease in contained chlorides, the indication is to awaken their action and proper secretion as rapidly as possible. Saline irrigations or injections, administration of salt in the food, supplies as far as may be at times two formal objects of treatment. When these fail we may give, by the mouth in proper form and dose, some intestinal antiseptic drug like betanaphthol, the salts of bismuth, creosote, charcoal, or a mineral acid, and obtain a good result.

Upon such medication in like case, I confess, I place but small reliance, in view of the evidences in all organs and functions of the intense and very grave toxæmia of the pneumonic infection.

A final word in regard to the use of alcohol in pneumonia. To my mind there can be little doubt that acute lobar pneumonia is one disease at least in which the use of alcohol is almost imperative at times if we wish to tide the patient over an imminent crisis. The frequency and amounts given at such times are governed almost solely by the urgency of the case and the apparent benefit derived. Under other conditions, especially with those unaccustomed to the use of alcohol when well, the amount given may be small or *nil*. With alcoholic subjects, of course, alcohol must be allowed freely, even though the disease may not be far advanced nor very threatening, except from our previous knowledge of how much such cases are always grave. As a matter of great practical importance, I would insist that the whiskey, brandy, or champagne which is taken by the patient should be the best or purest procurable. I am sure many cases of nausea or more pronounced stomachal intolerance, particularly in hospital treatment, are caused by cheap and bad spirits given to the patients. Can anything be worse than such symptoms at a time when the circulation needs all available extra stimulation? And is it not deplorable to think that these are caused or aggravated frequently by meanness or ignorance which permits or upholds the purchase of liquors for the treatment of the ill, of a very inferior grade, and which, in my judgment, are often injurious rather than beneficial.

In the treatment of no disease are frequently repeated visits from the physician, supported from the beginning with good nursing, more important than in that of pneumonia. In no disease, therefore, is free and unstinted expenditure of money more serviceable. Attention to these requirements will be the means of saving many lives which otherwise would be surely lost.

In conclusion, I wish again to emphasize what I regard as essential propositions. These are:

1. To begin judicious, rational treatment immediately and to continue it during the attack.

2. The most useful *single agent* in treatment, as preventive and curative, is creosote, used preferably as inhalations, properly given and continued for a sufficient length of time.

3. Strict avoidance of extremes of treatment in any direction, whether it be toward the use of so-called specifics or the employment of certain drugs, notably digitalis and strychnine.

4. It should be graven on our minds that pneumonia may be throttled or minimized most surely in the beginning. Later, when the disease is fully developed, our role is inferior, but should consist mainly in doing least harm.

5. Harm proceeds almost invariably from ignorance or undue enthusiasm.

CEREBRAL SKIAGRAPHY.[1]

By G. E. Pfahler, M.D.,

of Philadelphia,

formerly assistant chief resident physician in the Philadelphia Hospital; assistant physician and skiagrapher to the Medico-Chirurgical Hospital; lecturer upon medical terminology and symptomatology and demonstrator of physical diagnosis in the Medico-Chirurgical Hospital.

The Secretary has invited me to read a paper upon the above subject, and for this purpose I have selected 30 skiagraphs from a series of about 100 brain examinations, and have had lantern slides made for demonstration. The lantern slides, like the prints, do not show the detail that is found in the negative.

Some of the examinations have been made upon the cadaver and some upon the living subject.

I have omitted the clinical symptoms and observations. I have done this partly for the sake of brevity and partly because the histories have been or will be published elsewhere by the physicians in attendance.

My first case was a woman, aged thirty-two years, in the Philadelphia Hospital, in which Dr. Charles K. Mills had located a brain tumor by clinical symptoms and observations.[2] Through the kindness of Dr. Mills I was permitted to make an *x*-ray examination, with the idea of testing its value as a means of diagnosis.

The site of the tumor was located clinically in the left parietal region, possibly invading the motor region, and extending to or compressing the posterior limb of the internal capsule and the optic radiations where they approach one another in the region of the basal ganglia.

The left side of the head was therefore placed upon the plate, and the anode of the tube was placed directly opposite to the point

[1] Read before the annual meeting of the American Roentgen Ray Society, at St. Louis, September 8–13, 1904.

[2] Philadelphia Medical Journal, February 8, 1902.

at which Dr. Mills had located the tumor. An exposure of four minutes was made with a current of about six ampères and a tube, the vacuum of which corresponded to about two and one-half inches of air gap.

A negative was obtained which showed good detail of all the structures, namely, the scalp, the outer table of the skull, the diploë, and the inner table, the frontal, ethmoidal, sphenoidal, and mastoidal cells, the coronal suture, the groove of the posterior meningeal artery, the outline of the base of the skull, and shadows corresponding to the depressions for the frontal convolutions, and a large shadow, lying between the coronal suture and the posterior branch of the middle meningeal artery, corresponding to the area in which Dr. Mills had located the tumor.

For comparison, a colored woman of the same age was then selected, and a similar examination of the brain and skull made. This negative showed the normal structures, but did not show the shadow of the tumor seen in the negative of the first case, but did show diffuse shadows throughout the cerebral portion. These can probably be accounted for by the thickness and density of the skull, which is indicated by the thickness of the outer table, which is shown, and by the absence of all of the sutures and meningeal grooves. The absence of these lines is not due to a poor negative, as is shown by the fact that good definition is obtained of the structures at the base of the skull.

I then confirmed these observations by repeating the examination in each patient.

An operation was decided upon and performed by Dr. Hearn and Dr. Da Costa. They removed the part of the tumor corresponding to the upper portion of the shadow. The remainder was removed at autopsy, and was found to be subcortical and corresponded to the remainder and less dense part of the shadow.

At the suggestion of Dr. Mills, I then made some experiments upon the cadaver. The first subject was a man, aged sixty-three years. I hoped to show that a tumor could be shown in any of the usual locations. The usual incision for the removal of the scalp was made, and the left half of the cranial vault removed, leaving the right intact. Without disturbing the brain, and carefully replacing the section of bone and suturing the scalp, I made a negative, to show the normal shadows of this particular skull and brain (Fig. 1). I then removed a section of brain corresponding to a hardened cerebellar tumor, the specimen being from a case reported by Dr. J. Hendrie Lloyd. I placed the tumor in the cavity made for it and replaced the section of bone and sutured the scalp, so as to preserve as nearly as possible the conditions that would be found in the living subject. The negative made showed a very distinct shadow of the tumor.

The tumor was removed from this site and placed in the frontal

region, having for its boundary the first left frontal convolution, and for its posterior boundary the central fissure. The brain tissue was replaced in the first site and a negative made which showed the tumor, but with less dense shadow than in the first position. This was probably due to a more penetrating light. This negative showed the absence of brain tissue where it had not been cut to fit the tumor exactly.

FIG. 1.

Normal skull and brain.

The tumor was now placed in the posterior portion of the left hemisphere, the frontal section replaced, a negative made, and the skull and scalp closed as before. This negative not only showed the tumor, but also the outline of the section of brain in the frontal region which had been replaced.

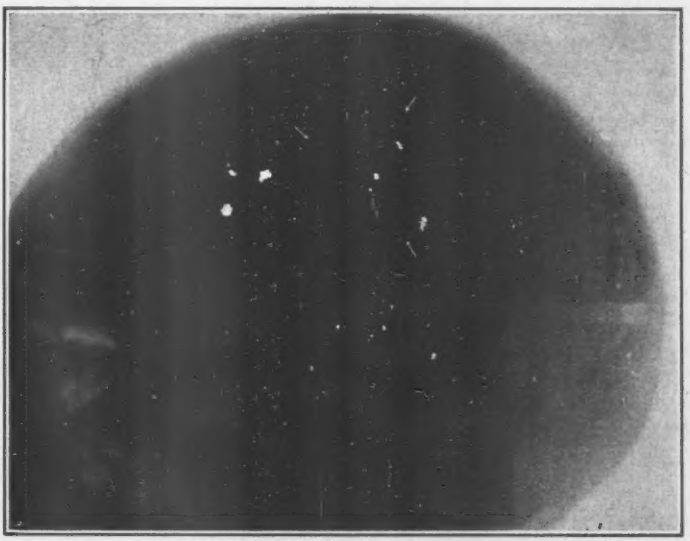
Dr. Mills and Dr. Spiller reminded me that a hardened tumor in a fresh brain might not be a fair test. To harden it the brain was placed in a 10 per cent. formalin solution for fifteen hours. It was then returned to the skull and the tumor replaced in the motor region. A good shadow of the tumor was obtained, and the markings of each section of the brain tissue previously made were shown (Fig. 2).

My second subject was a white woman, aged twenty-nine years. In this series I hoped to show that a cerebellar tumor could be

shown, and also that the different varieties of tumors could be photographed. I used all the free tumors obtainable. The first was a cerebellar sarcoma from a case studied by Dr. James Hendrie Lloyd, the specimen being preserved in the Pathological Museum of the Philadelphia Hospital.

The tumor was placed as nearly as possible in the same position as it had been found in Dr. Lloyd's case. A definite shadow of the tumor was obtained. I then used a perithelioma which had been removed from a case of Dr. Mills. The tumor was skiagraphed in the central part of the motor area. The next was an endothelioma

FIG. 2.



Location of tumor is indicated by the arrows. See location of the cuts into the brain tissue, in the posterior frontal region, there being no actual loss of tissue.

loaned by Dr. Spiller. This was also placed in the motor area and a shadow obtained of only slightly greater density than that of the surrounding brain tissue, but the sections of the tumor itself were shown. In another experiment a large spindle-celled sarcoma of the pineal gland was placed in the motor region and a shadow obtained.

In all of these series the plates were overexposed, though the time had only been four minutes in each case. I then repeated the series upon the body of a man, aged fifty-four years, timing the plates three minutes, with the result that all were underexposed. Still feeling that my results might be questioned, because I had used

a hardened tumor in a fresh brain, I used the following test and repeated it: Half of a fresh brain was taken and placed beside the half of a hardened brain and the two photographed with the hardened tumor on the same plate and under the same light. The hardened brain was a little larger than the fresh one. The negative showed more detail in the hardened brain, but the density of the shadow was little if any greater than that of the fresh one. The shadow of the tumor was more dense than either one.

Realizing that much of the success in skiagraphy of the brain must of necessity depend upon accurate technique, I therefore conducted a series of experiments to find the best possible technique. In skiagraphy we have five principal varying factors. These are the distance of the anode from the plate, the time of exposure, the vacuum of the tube, the amount of current, and the thickness and density of the part examined. The first four are more or less under our control. The fifth is always variable, and, therefore, the other four must be judged and regulated in proportion to the fifth. Fortunately, the variability in the thickness of the adult skull is not very variable, but must not be entirely ignored.

In this series of experiments I used at one and the same time three different tumors, one a spindle-celled sarcoma, one an epithelioma, and one a perithelioma. One was placed in the prefrontal region, one in the motor region, and one between the two halves of the cerebellum. My first series of plates was made to determine the best time of exposure. In this test I used a tube the vacuum of which was equal to two and one-half inches of parallel air-spark at a distance of eighteen inches, and as the subject I had the body of a woman, aged forty-two years. The time of my exposures were two, two and one-half, three, three and one-half, and four inches, respectively.

My best results were obtained with an exposure of three and one-half minutes.

I then made a series of negatives in which the distances of the anode from the plate were twelve, fifteen, eighteen, and twenty-one inches, respectively. My best results were obtained at a distance of eighteen inches. At this distance the shadows of the proximal side of the skull will be more or less diffused, and yet sufficient detail of the opposite half of the brain be obtained.

The degree of vacuum is our most variable factor and the one that is least under our control, and, therefore, experience and judgment in varying the other factors accordingly will have an important bearing upon the results in each case. I used a self-regulating tube and used as nearly as I could vacuums corresponding to two, two-and-one-half, three, and three-and-one-half inches of parellel air-spark.

My best results were obtained with a vacuum of two-and-one-half inches.

The developer that I used was the following:

A.—Metol	4 drachms.
Hydrochinone	4 "
Sodii sulphite	5 ounces.
Water	5 pints.
B.—Sodii carbonate	4 ounces.
Water	30 "

Use two parts of A. and one of B.

These experiments were made about three years ago, while I was at the Philadelphia Hospital. At that time I was using a fifteen-inch coil with a mechanical interrupter, and used a current of 8 to 10 ampères. With our modern technique and modern coils the time must be reduced. At the present time I make my exposure one and one-half minutes, with 8 to 10 ampères and the mechanical interrupter, or in forty-five seconds with the electrolytic interrupter and 25 ampères.

Since the above experiments I have had the opportunity of making a fairly large number of brain examinations. Most of them were obscure cases. Several of these are worthy of mention.

One, a case of Dr. Charles K. Mills,[1] in which the *x*-ray examination showed the normal details of the skull, and, in addition, showed an abnormal shadow about three inches in diameter and irregular in outline lying directly in the motor region. This was interpreted as the shadow of the tumor, and corresponded to the clinical diagnosis. The shadow of the inner table of the skull was seen to be interrupted in the region of the tumor and seemed to be disorganized. At the time of the operation, by Dr. John B. Deaver, the inner table was found to be diseased and gave rise to considerable hemorrhage. The tumor was removed and found to correspond in size and position to the shadow in the skiagraph.

In another case of Dr. Mills,[2] which was of unusual interest, the skiagraphic examination showed the shadow of a gumma in the motor region of the left side of the brain. The shadow was two and one-half inches in length, one and one-half inches in width at its widest portion, irregular in outline, and, from its density, I estimated it to be about one-half inch in thickness. The gumma when removed by Dr. Hearn and Dr. Da Costa corresponded exactly to the location and dimensions given in the skiagraphic examination (Fig. 3).

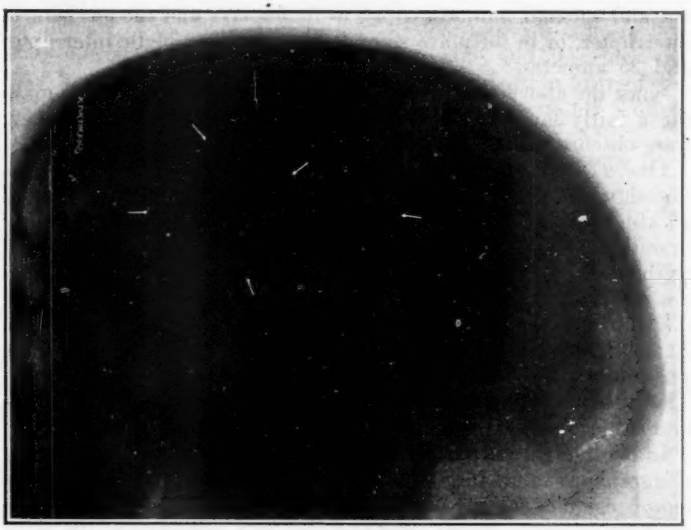
There are several other cases of brain tumor in which the diagnosis has not been decided by either operation or autopsy, and, therefore, of little value scientifically. One of these is a case referred by Dr. C. A. Veasey for *x*-ray examination.[3] He had suspected a tumor of the pituitary body, because of bitemporal hemianopsia.

[1] Philadelphia Medical Journal, September 27, 1902.
[2] Ibid., November 29, 1902.
[3] Transactions of the American Ophthalmological Association, 1904.

The skiagraph showed a slight shadow, about one-half inch in diameter, and located in the region of the pituitary body. This shadow must, of course, only be accepted as confirmatory, and not in itself diagnostic. In fact, if it did not agree with the clinical symptoms, I should ignore it, for such a small tumor located in the central portion of the brain could not be expected to cast a definite shadow.

We sometimes learn more from our failures than from our successes, and, therefore, I desire to mention an instance in which I was mistaken recently. The case will be reported in detail later, and, therefore, I shall only note it in so far as it has a bearing upon

FIG. 3.

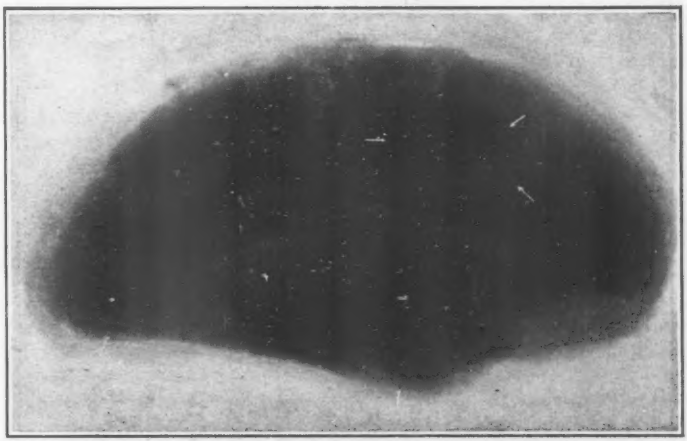


Case of Dr. Mills. Gumma indicated by the arrows.

the subject under consideration. The case was referred by Dr. B. F. Hawley and Dr. Judson Daland. A brain tumor was suspected, but the symptoms were obscure, and no definite diagnosis could be made. The skiagraph showed a large shadow occupying in part the region of the cerebellum and in part the posterior portion of the cerebrum. An operation was performed by Dr. W. W. Keen, and no tumor was found. Autopsy showed no tumor in this region, but showed a tumor, about one and one-quarter inches in diameter, lying farther anteriorly. The autopsy was performed by Dr. W. G. Spiller, at which I was priviledged to be present. There were several other very small tumors found, but nothing was found

either in the skull nor in the brain to account for the shadow that I had obtained. I was very much disappointed and puzzled. I believe, however, that this shadow was produced by the greater diameter of the head at this point and by the contact of the head at this point which I find interferes with the developing. Allowance must always be made for these shadows. This was only half of an explanation, since it did not account for the fact that I did not find the tumor where it was located. Through the kindness of Dr. Spiller, I was permitted to examine the brain skiagraphically. I made several lateral and vertical views. Beautiful details of the lobes and convolutions and of the ventricles of the brain were

FIG. 4.



Case of Drs. Hawley and Daland. Skiagraph of the brain, post-mortem. Tumor indicated by the arrows.

obtained. The shadow of the tumor could be recognized, but the density of this shadow was so little greater than the surrounding brain tissue that I feel sure it would have been impossible to locate it skiagraphically while in the skull. The skiagraphic examination may still be of value in distinguishing this variety of tumor from others (Fig. 4).

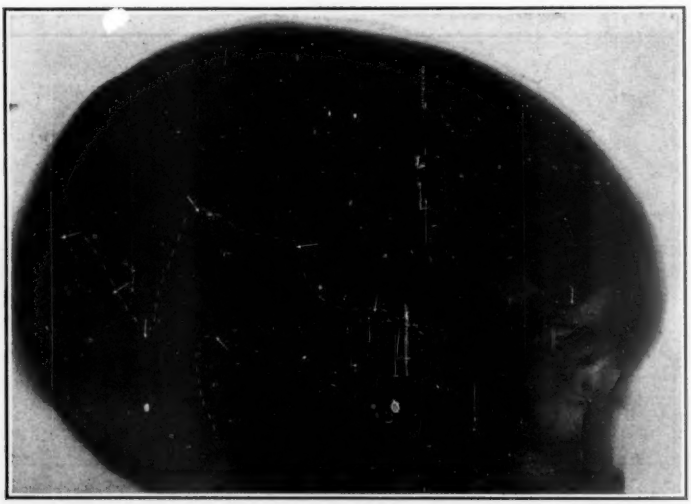
In addition to the brain tumors studied, I have had the opportunity to examine three cases of softening of the brain. Two of these were examined post-mortem, and one was examined during life and the diagnosis confirmed by autopsy.

The first was Dr. Charles W. Burr's case.[1] The case was one of

[1] THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, February, 1904.

thrombosis of the midcerebral artery, with cystic degeneration, and causing aphasia and hemiplegia. The examination was made post-mortem. The brain was replaced and the skull and scalp closed. I then made a negative of the affected side and also of the opposite side, because I believed that possibly the normal side could be compared with the affected sides. This case, however, demonstrated that this cannot be relied upon, for the lesion was shown upon both negatives, but with much more definite outline on the affected side. The skiagraph showed above the cerebellum and petrous portion of the temporal bone a light area which corresponded exactly with

FIG. 5.



Dr. Burr's case. Area of softening is indicated by the arrows.

the outline of the area of degeneration. This skiagraph showed, also remakably well the convolutions of the cerebellum (Fig. 5).

The second case was also one of Dr. Burr's, in which an irregular area of degeneration was found in the distribution of the midcerebral artery and which had caused hemiplegia. The skiagraph showed transparent areas which corresponded exactly with the area of degeneration (Fig. 5).

The third case was a very interesting case of aphasia, in the Insane Department of the Philadelphia Hospital, which was studied very carefully by Dr. Charles K. Mills.[1] Two skiagraphs made at intervals of several months showed a light area about one inch in diam-

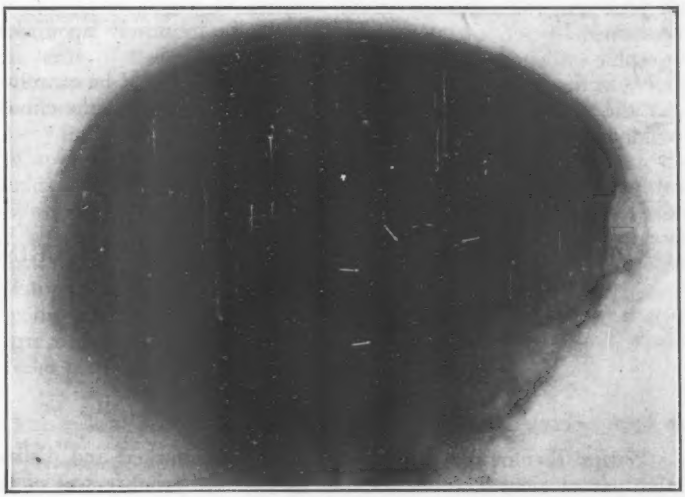
[1] THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, September, 1904.

eter (Fig. 6), located in the region of the speech centre. The autopsy showed a corresponding area of softening involving the posterior portion of the third left frontal convolution and the island of Reil.

Some of the results obtained in skiagraphy of the brain have been most surprising and gratifying both to myself and others. Other results have been most disappointing.

Dr. Church, of Chicago, records a case of cerebellar tumor in which the *x*-rays were used by Mr. W. H. Fuchs. Skiagraphs of the tumor were obtained. At autopsy a highly vascular gliomatous

FIG. 6.



Dr. Mills' case. Area of softening is indicated by the arrows.

tumor was found, the tumor being the seat of several old and recent hemorrhages, and also of a recent clot of considerable size.[1]

Obici and Ballici[2] demonstrated, by means of the *x*-ray the presence of tumor in a case of a boy who died of brain tumor, the experiment being performed post-mortem. They also experimented with tumors of different kinds placed in the brain of cadavers, and were, in some instances, able to obtain localizing shadows.

Oppenheim,[3] in reviewing the subject of brain tumors in his text-book, says that his attempts to use the *x*-ray for diagnostic purposes

[1] THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, February, 1899.

[2] Rivista di Patholog, October, 1897. Cited by Church.

[3] Diseases of the Nervous System. Translation by Edw. Mayer, M.D., Philadelphia and London, 1900.

failed, although he was able to determine that a tumor placed within the cranium upon the brain was very distinctly noticed.

Pancoast and McCarthy conclude that the value of the Roentgen rays in brain lesions is at present dubious.[1]

In addition to the tumors and cases of softening, I have had a case in which the skiagraph seemed to show thickening of the membranes to a considerable extent. This corresponded to the clinical symptoms. No autopsy has been obtained, and, therefore, I can draw no definite conclusion.

In conclusion, I believe that we should be able to show in the skiagraph most large lesions, such as new-growths, softening, hemorrhage, and abscess; but I want to repeat what I said at our meeting a year ago, when discussing this subject, "I would never take the responsibility of an operation upon the brain purely upon skiagraphic evidence.

"On the other hand, all of the above cases should be examined by the *x*-ray, with the idea of confirming or adding to the clinical evidence."

ON THE PRESENCE OF ORGANIC ACID IN THE URINE IN CASES OF RHEUMATOID ARTHRITIS.

By Helen Baldwin, M.D.,
of New York.

(From the Laboratory of Dr. C. A. Herter, New York.)

While rheumatoid arthritis presents such marked and distinctive pathological features, its etiology and the primary seat of the affection are still unknown. Because of this the study of the metabolic changes taking place in the course of the disease becomes of especial interest, and any observation seemingly of small moment may prove of real value in helping to determine the cause of the morbid condition.

It is with this in view that the following observations are recorded, relating to the presence of an organic acid in the urine of patients suffering from this disease.

From the time of the first examinations of urinary sediment with the microscope, the presence of certain organic acids in the urine has been recognized. Uric acid, oxalic acid, and hippuric acid, by their precipitation under morbid conditions, have been forced upon the attention of pathologists, and in the study of methods for their

[1] University of Pennsylvania Medical Bulletin, March, 1903.

estimation it was early found that they were present in the urine in solution even when not found in the sediment. It was not, however, until the study of acid intoxications by Gaethgens,[1] Walter,[2] Salkowski,[3] and later others, as Stadelmann[4] and Magnus-Levy,[5] that methods were discovered for detecting organic acid in the urine and approximately estimating the amount present even when the nature of the acid was not known.

The method employed by Magnus-Levy[6] in determining the amount of β-oxybutyric acid excreted in diabetes is the one which has been used in making the following observations. This method is based upon the fact which has become well established that acids are excreted from the kidneys, not as free acids, but only in combination with bases to form salts. This being the case, if all the acids and bases present in the urine are estimated and represented in terms of their chemical equivalents, with reference to the same substance as sodium, the total acid present will be found to exactly balance the sum of the bases. Gaethgens, Hagentorn, and Magnus-Levy found that when the known acid and basic constituents present in appreciable quantities in normal urine were thus estimated, the acids and bases did almost exactly balance each other. There was, however, uniformly present a slight excess of acids over the bases determined, and this was considered to be balanced by organic bases such as the purin bodies, which had not been estimated. Estimations were made of potassium oxide, sodium oxide, calcium oxide, magnesium oxide, and ammonia, and of hydrochloric acid, uric acid, preformed sulphuric acid, combined sulphuric acid, monobasic phosphoric acid, and dibasic phosphoric acid.[7]

[1] Zur Frage der Ausscheidung freier Säuren durch den Harn. Med. Centralbl., 1872, S. 833. Ueber Ammoniakausscheidung, Zeitschr. f. physiolog. Chemie, Bd. iv. S. 35.

[2] Untersuchungen über die Wirkung der Säuren auf den thierischen Organismus, Archiv f. experiment. Pathol. u. Pharmakol., 1877, Bd. vii. S. 148.

[3] Ueber die Möglichkeit der Alkalienziehung am lebenden Thiere, Virchow's Archiv, 58, S. 1.

[4] Ueber den Einfluss der Alkalien, u. s. w., Stuttgart, 1890, S. 122 ff. bei Hagentorn.

[5] Die Oxybuttersäure und ihre Beziehungen zum Coma diabeticum, Archiv f. experiment. Pathol. u. Pharmakol., 1899, Bd. xlii. S. 149. Untersuchungen über die Acidosis im Diabetes mellitus und die Saureintoxication im Coma diabeticum, Archiv f. experiment. Pathol. u Pharmakol., 1900–1901, Bk. xlv. S. 388.

[6] For a detailed discussion of this method reference is made to Magnus-Levy's article, loc. cit.

[7] The following methods were employed in estimating the acids and bases. (See Neubauer und Vogel's Harnanalyse.)

Potassium and sodium by Lehmann's method. Separation by indirect volumetric method.

Calcium oxide, by precipitating as CaC_2O_4 and weighing as CaO.

MgO, by precipitating as NH_4MgPO_4 and weighing as $Mg_2P_2O_7$.

NH_3, by Schlösing's method.

SO_3 preformed and SO_3 combined, by Salkowski's gravimetric method.

P_2O_5 total, by Neubauer's volumetric method.

P_2O_5 dibasic and monobasic, by Freund's method.

Cl, by Mohr's method.

Uric acid, volumetrically by Folin's method. Zeitschr. f. physiolog. Chemie., Bd. xxxii. S. 514.

The following cases from our records illustrate the balancing of acids and bases in twenty-four-hour specimens of normal urine.[1] The weights are given in the sodium equivalent of each acid or base (Table I.):

TABLE I.

Case	I.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.	X.
K_2O	2.525	1.547	2.5720	1.0470	0.7286	1.2840	0.9232	1.6360	2.3740	0.9757
Na_2O	2.001	4.249	5.2250	2.4040	1.3300	2.1090	3.881	3.7040	4.0930	1.2970
CaO	0.2165	0.476	0.2289	0.0126	0.0768	0.0722	0.2154	0.2319	0.0952	0.0307
MgO	0.1439	0.268	0.1302	0.1298	0.0664	0.1757	0.0806	0.1733	0.2237	0.1098
$N(H_3)$	0.6127	1.398	1.8910	0.3186	0.2859	0.4561	0.4704	1.5480	1.0930	0.5181
Total bases	5.4991	7.938	9.5471	3.9070	2.4877	4.0970	5.5706	7.2882	7.8789	2.9813
SO_3 preformed	0.9051	1.420	1.5390	0.5584	0.4709	0.7078	0.9734	1.4500	1.2880	0.6570
SO_3 combined	0.0465	0.073	0.1347	0.0219	0.0150	0.0167	0.0356	0.0564	0.0663	0.0243
P_2O_5 dibasic	1.300	0.782	1.1930	0.5864	0.4808	3.0730	0.7432	0.8680	0.7387	0.4212
P_2O_5 monobasic	0.0849	0.601	0.3023	0.0408	0.0548	0.0364	0.1987	0.3715	0.2916	0.2106
Cl	3.171	5.386	6.9720	2.8280	1.5180	0.5994	4.476	4.5910	5.4130	1.6360
Uric acid	0.0432	0.117	0.0894	0.0221	0.0143	0.0930	0.0584	0.0911	0.0870	0.0431
Total acids	5.5507	8.379	10.2304	4.0576	2.5538	4.5263	6.4853	7.4280	7.8846	2.9922
Apparent excess of acids balanced by organic bases	0.0516	0.441	0.6833	0.1506	0.0661	0.4293	0.9147	0.1398	0.0057	0.0609

If, now, an acid is excreted in the urine other than those ordinarily found its presence can be detected in estimating the acids and bases, for in this case there will be an excess of bases over the acids normally present, the excess being required to neutralize this acid, which is not normally excreted. This may be illustrated by a twenty-four-hour specimen of urine from a case of diabetes mellitus. The patient had 5 per cent. of sugar in the urine.

The analysis is as follows, the weights as before being in equivalent weights of sodium:

K_2O	5.0070 grms.	SO_3 preformed	1.5950 grms.
Na_2O	6.5030	SO_3 combined	0.0566
CaO	0.2051	P_2O_5 dibasic	2.0160
MgO	0.3910	P_2O_5 monobasic	0.5054
$N(H_3)$	1.7120	Uric acid	0.1719
		Cl	6.5030
Total bases	13.8181	Total acids	10.8579

The excess of bases over the acids estimated in this case is 2.9602 grams, or 21 per cent. of the total bases; that is, the amount of organic acid present (in this case known to be chiefly β-oxybutyric acid) is that which will combine with 2.96 grams of sodium.

[1] It is, of course, necessary to use the greatest care in preventing any decomposition of the specimens, since the conversion of urea into ammonium carbonate will invalidate the result. The separate portions of urine should be thoroughly shaken with carbolic acid, chloroform, or thymol before any bacterial action can begin.

It is also evident that if any organic acid, as salicylic acid or the salicylates, were administered at the time of the observations, it would be estimated with the other organic acid excreted.

Magnus-Levy used this method chiefly in estimating the amount of organic acid present in diabetic urine. He mentions, however, that organic acid is in this way found to be present in lesser amounts in other conditions, such as starvation, intestinal disturbance, phosphorus poisoning, acute yellow atrophy, and fever. Herter and Wakeman,[1] using Magnus-Levy's method of analysis, reported the finding of organic acid in the urine in cases of dilatation of the stomach, with atony, rheumatoid arthritis, and cirrhosis of the liver.

Since in the cases of rheumatoid arthritis reported by Dr. Herter there was found organic acid in the urine other than that normally present, it has seemed well to continue this investigation. I have, therefore, estimated the acids and bases in the urine in all cases of this kind as patients happened to come under observation.

The following records include reports of forty specimens of urine from twenty-one different patients. In all cases in which the disease was progressing—that is, in thirty-three of the specimens—there was found to be an excess of the bases over the acids normally present, thus indicating the presence of organic acid. The remaining seven specimens were from patients in whom the symptoms were stationary or improving, and in these the analysis showed a normal condition—that is, with the estimated acids slightly in excess of the bases.

In studying these records it should always be borne in mind that an apparent excess of bases indicates the presence of a corresponding amount of some acid not normally present in the urine, and so not estimated.

CASE I.—An illustrative case may be reported in detail.[2] The patient was a young woman who for several years had been suffering from rheumatoid arthritis. Both ankles, both knees, both wrists, and the small joints of the hands were involved, with swelling, tenderness, pain, deformity, and impairment of motion. At the time that the first analyses were made the disease was steadily progressing. The results of the urinary analysis were as follows:

October 26, 1899.	K_2O	1.3370	SO_3 preformed . . .	0.9961
	Na_2O	3.9780	SO_3 combined . . .	0.0388
	CaO	0.1345	P_2O_5 dibasic	0.6725
	MgO	0.1861	P_2O_5 monobasic . . .	0.1400
	$N(H_3)$. . . .	0.9663	Uric acid	0.0868
			Cl	3.8830
	Total bases .	6.6019	Total acids . . .	5.8172

Excess of bases 0.7847, or 11.9 per cent. of total bases.

[1] Notes on Acid Intoxications. Proceedings of New York Pathological Society, 1900–1901. C. A. Herter. The Pathology of Acid Intoxications. Transactions of the Association of American Physicians, vol. xv.

[2] Only a brief summary of the condition of these cases is given, since the question as to whether the disease is progressive or stationary seems to be the one determining whether or not the abnormal acid is present in the urine.

27th. Total bases, 4.7823; total acids, 4.6475; excess of bases, 0.1348; 2.8 per cent. of total bases.

28th. Total bases, 7.9051; total acids, 7.2755; excess of bases, 0.6296; 7.8 per cent. of total bases.

January 20, 1900. Total bases, 5.7926; total acids, 5.1932; excess of bases, 0.5994; 10.3 per cent. of total bases.

At this time the stomach contents were examined, and there was found to be no hydrochloric acid present after a test meal, although there had been no gastric symptoms. Lavage was used about twice a week for the next ten months. At first the stomach contents were always fermented and contained fatty acids, but after routine washings for several months hydrochloric acid was found in the stomach after a test meal.

Urinary Examination. February 14, 1900. Total bases, 6.7765; total acids, 6.1873; excess of bases, 0.5892; 8.7 per cent. of total bases.

April 4th. Total bases, 6.8089; total acids, 6.3930; excess of bases, 0.4159; 6.1 per cent. of total bases.

May 14th. Total bases, 7.2896; total acids, 6.9305; excess of bases, 0.3591; 4.9 per cent. of total bases.

The patient spent the winter of 1900 and 1901 in California, living in the open air and exercising on horseback, with no other treatment. The next analysis was after a year's interval

May 14, 1901. Total bases, 8.0026; total acids, 8.1500; excess of *acids*, 0.1474. At this time the symptoms were all in abeyance. There remained some deformity of the joints and some weakness in the muscles, but the disease was for the time arrested in its course.

CASE II.—Another case in which the amount of the abnormal acid was diminished in the urine, with an improvement in the other symptoms, is the following:

The patient was a working girl who had for several years been suffering from increasingly severe symptoms in the joints of the wrists, fingers, knees, and ankles, with characteristic deformities in the joints. She could no longer work, and she could with difficulty walk about.

The stomach contents were examined after a test meal, and there was absence of hydrochloric acid.

Urinary Analysis, February 19, 1900. Total bases, 5.0916; total acids, 4.4570; excess of bases, 0.6346; 12.4 per cent. of total bases.

The patient spent the eight months following this examination under as favorable hygienic surroundings as were possible for a person of small means.

She took hydrochloric acid after meals throughout this time. She markedly improved in health, and became able to use her hands and feet, with but little discomfort.

Urinalysis, November 8, 1900. Total bases, 4.3337; total acids, 4.1108; excess of bases, 0.2229; 5.1 per cent. of total bases.

CASE III.—Another patient showing an improvement in symptoms which was not as marked as in the case of those cited above was the following: On May 24, 1900, the patient was first seen. She then complained of pain in the shoulders, knees, and soles of the feet, with deformity of the hands. Two years before the patient had begun to have severe pain in the hands, extending to the arms. She was unable to sleep on account of the pain, and often walked the floor at night. Then pain developed in the soles of the feet, in the knees, and in the legs. Not until a year later did the joint swelling in the hands begin. There was steadily increasing disability from this time. On examination the hands were found to be in a position of extension and ulnar deflection. Flexion is much limited, extension nearly complete. The lower ends of the ulnar bones are very conspicuous and bulbous. The carpometacarpal joints of the hands are most involved. The right knee is swollen, and there is apparently extra-articular thickening. Extension of this knee is painful. The left knee is not swollen, but is painful on motion. There is no joint deformity in the foot except hallux valgus.

June 7, 1900. The stomach contents, examined after a test meal, showed absence of hydrochloric acid. Total bases in the urine, 5.5391; total acids, 4.4402; excess of bases, 1.0989; 19.8 per cent. of total bases.

The patient improved slowly throughout the summer, but still had a good deal of tenderness in the affected joints when the next examination was made.

December 14*th*. Total bases, 3.0073; total acids, 2.9989; excess of bases, 0.0084; 0.28 per cent. of total bases.

CASE IV.—The following record is that of a patient who was first seen in an interval when she was having no active symptoms. Three years before this she had had rapidly developing symptoms from the disease, with involvement of the knees, ankles, elbows, wrists, and the metacarpophalangeal joints of both hands. At the time when the first urinary examination was made she had remaining some deformity of the joints in the hands, with limitation of motion in the right wrist and left ankle. The urine was at this time normal as regards the balancing of acids and bases. Total bases, 5.0979; total acids, 5.2541; excess of *acid*, 0.1562. Two years after this there was an exacerbation of the symptoms, brought on by exposure to cold. All the joints involved were affected, with very severe pain in the knees on standing and rapid atrophy and loss of power in the muscles. Total bases in twenty-four-hour specimen, 7.9067; total acids, 7.6305; excess of *bases*, 0.2762; 3.5 per cent. of total bases. Five months later, when the acute symptoms were again subsiding, the urine was again normal. Total bases, 4.7278; total acids, 5.1523; excess of *acid*, 0.4245.

The following records are from fifteen cases in which the disease was progressing. It will be noted that in all of these there is an excess of bases over the acids estimated (Table II.):

TABLE II.

	Case V.			Case VI.				Case VII.		Case VIII.	
	Nov. 12, 1900	Dec., 1900	Jan. 31, 1901	Jan. 26, 1900	Jan. 31, 1900	Feb. 21, 1901	1901	Jan. 10, 1900	Apr. 30, 1900	Feb. 11, 1902	Jan. 17, 1903
K_2O	1.1060	0.8600	1.0340	1.027	0.8367	0.9879	0.6258	2.651	2.1200	0.7816	0.5119
Na_2O	2.2730	2.2472	2.4010	1.136	1.2730	0.8217	0.8453	1.013	0.4369	1.992	1.413
CaO	0.1232	0.1290	0.0709	0.1227	0.1039	0.1035	0.1070	0.0605	0.2032	0.0613	0.0821
MgO	0.1915	0.0753	0.0524	0.0350	0.0751	0.0764	0.0903	0.1496	0.1151	0.0814	0.0232
$N(H_3)$	0.4816	0.6272	0.3238	0.6516	0.2541	0.4374	0.2462	0.5401	0.5967	1.298	0.6105
Total bases	4.1753	3.9387	3.8811	2.9723	2.5428	2.4269	1.9146	4.4142	3.4719	4.2143	2.6407
SO_3 preformed	0.4061	0.4949	0.1644	0.4239	0.8238	0.5624	0.3434	0.7177	0.5333	0.6798	0.5501
SO_3 combined	0.0373	0.0335	0.0310	0.0275	0.0305	0.0526	0.0452	0.0600	0.0278	0.0443	0.0455
P_2O_5 dibasic	0.4652	0.4017	0.3889	0.5459	0.1936	0.3286	0.2333	0.6385	0.1313	0.6125	0.9298
P_2O_5 monobasic	0.0259	0.1653	0.0583	0.0428	0.1571	0.2456	0.1846	0.0339	0.1804	0.1531	0.0211
Uric acid	0.0630	0.0420	0.0389	0.0472	0.0302	0.0372	0.0281	0.0433	0.0389	0.0257	0.0821
Cl	2.6120	2.123	2.5540	1.581	1.4870	0.9817	0.8962	2.8000	3.4620	1.954	0.8847
Total acids	3.6155	3.2604	3.2355	2.6683	2.2223	2.2081	1.7308	4.2934	3.3737	3.4694	2.5133
Excess of bases	0.5598	0.6783	0.6456	0.304	0.3205	0.2188	0.1838	0.1208	0.0982	0.7449	0.1274
Per cent. of excess	13.4	17.2	16.6	10.2	12.6	9.02	9.6	2.7	2.8	17.6	4.8

	Case IX.	Case X.	Case XI.	Case XII.	Case XIII.	Case XIV.	Case XV.	Case XVI.	Case XVII.	Case XVIII.	Case XIX.
K_2O	1.3550	1.3385	0.6175	0.7256	1.485	1.558	0.6947	0.9694	0.5495	1.2690	0.8768
Na_2O	3.4010	2.3930	0.9828	3.043	1.088	2.862	4.116	0.8768	3.299	2.1780	1.8980
CaO	0.1184	0.2412	0.2241	0.0634	0.0354	0.2558	0.0952	0.0768	0.1429	0.0965	0.0843
MgO	0.0885	0.1430	0.0746	0.1306	0.1144	0.2125	0.1312	0.0343	0.1432	0.1199	0.1044
$N(H_3)$	1.0440	1.0875	0.5753	0.9131	1.121	1.016	0.2669	0.8861	1.154	0.2226	0.4651
Total bases	6.0069	5.2032	2.4743	4.8757	3.8438	5.9043	5.3040	2.8434	5.2886	3.8860	3.4286
SO_3 preformed	0.6033	0.4935	0.5315	0.6656	0.7519	0.7794	0.2662	0.2433	0.7039	0.4888	0.3082
SO_3 combined	0.0757	0.0463	0.0390	0.0495	0.0372	0.0460	0.0380	0.0409	0.0563	0.0344	0.0321
P_2O_5 dibasic	0.6545	0.6740	0.2522	0.8435	0.6415	1.096	0.5834	0.3796	0.6834	0.6674	0.8085
P_2O_5 monobasic	0.0123	0.0114	0.0074	0.1061	0.1040	0.1801	0.0436	0.0000	0.3095	0.0458	0.0917
Uric acid	0.0586	0.0541	0.0586	0.0456	0.0407	0.0635	0.0391	0.0743	0.2386	0.0090	0.0198
Cl	3.8780	3.1405	1.0420	3.038	2.129	3.407	3.256	0.7676	3.126	2.1630	1.9110
Total acids	5.2824	4.4198	1.9307	4.7483	3.7043	5.5720	4.2263	1.5057	5.1127	3.4079	3.1658
Excess of bases	0.7245	0.7834	0.5436	0.1274	0.1395	0.3323	1.0777	0.8377	0.1759	0.4781	0.2628
Per cent. of excess	12.1	15	22	2.6	3.6	5.6	20.3	35	3.3	12.3	7.6

Case XVI., which shows the largest amount of organic acid, is that of a very marked case of arthritis deformans in which there was almost complete ankylosis of all the joints. The specimen was examined a few weeks before the patient's death. The urine was alkaline, but showed no evidences of decomposition.

In the two remaining cases, in each of which only a single urinary examination was made, the disease had become stationary. So

while there was much disability and rigidity of the joints, the disease was not progressive. The following records are from these patients (Table III.):

TABLE III.

	Case XX.	Case XXI.
K_2O	0.8461	0.6828
Na_2O	1.489	4.625
CaO	0.1138	0.4042
MgO	0.1695	0.1491
$N(H_3)$	0.6114	1.221
Total bases	3.2298	7 0816
SO_3 preformed	0.5099	1.261
SO_3 combined	0.353	0.0702
P_2O_5 diabasic	0.6234	0 2159
P_2O_5 monobasic	0.1266	0 7511
Cl	1.904	4.865
Uric acid	0.0421	0 0196
Total acids	3.2413	7.1828
Excess of acid.	0.0115	0.1012

ON THE OCCURRENCE OF INTESTINAL PUTREFACTION IN RHEUMATOID ARTHRITIS. While the analyses of these specimens of urine were made primarily to determine the relative amount of acids and bases present, there are certain other points of interest with reference to these cases.

Since the work of Baumann it has been recognized that the amount of intestinal putrefaction can be approximately measured by the ratio of the aromatic sulphates to the simple (preformed) sulphates excreted in the urine. Certain aromatic products of intestinal putrefaction, as indol, skatol, and phenol, become linked in the body with sulphuric acid to form the so-called ethereal or aromatic sulphates. Normally these bear a ratio to the simple preformed sulphates of from 1:10 to 1:15, but in cases of intestinal putrefaction the ratio increases to 1:8 or 1:5, or even in exceptional cases, so that the amount of aromatic sulphates is equal to that of the preformed sulphates.

In certain cases of intestinal putrefaction, however, one of these aromatic products may be formed in excess, while the total aromatic sulphates will not be above the normal. Thus the presence of indican in the urine, indicating the formation of indol by a putrefactive process, may be noted in a given case in which the total ethereal sulphates are in normal amount, or the reverse may be true, that indican may be entirely absent from the urine while the formation of other products of putrefaction leads to an excess of aromatic sulphates. In all but two of the cases of rheumatoid arthritis noted above, in which the disease was progressing, there was found either an excess of aromatic sulphates in the urine or else the presence of indican, and these two were mild cases.

In twenty-three specimens there was an increase in the aromatic sulphates excreted, the ratio varying from 1:2.6 to 1:9.8, the average being 1:6.2 (Table IV.):

TABLE IV.

	Case II.		Case III.		Case IV.			Case V.		
	Feb. 19, 1900.	Nov. 8, 1900, improved.	June 7, 1900.	Dec. 14, 1900 improved.	Nov. 15, 1000, no active symptoms.	July 5, 1902, exacerbation.	Nov. 23, 1902, improved.	Nov. 12, 1900,	Dec., 1900.	Jan. 31, 1900.
Volume	1260	740	1400	567	1040	1500	980	1080	900	1120
Specific gravity	1017	1026	1013	1023	1020	1021	1024	1015	1016	1011
Reaction	acid	st'ngly acid	acid	acid	st'ngly acid	acid	st'ngly acid	sl'ghly acid	acid	sl'ghly acid
Albumin	0	0	0	0	0	0	0	0	0	0
Indican	0	...	...	0	0	0	0	...	0	present
Urea	20.03	16.42	22.48	12.17	26.92	33.06	28.4	12.27	14.19	9.85
Uric acid	0.1955	0.4047	0.2072	0.219	0.5618	0.9214	0.5373	0.460	0.3063	0.283
Ratio $\frac{\text{urea}}{\text{uric acid}}$	102	41	108	55	48	36	44	27	46	35
H_2SO_4 preformed	1.7151	1.6063	1.260	1.047	2.068	2.858	1.647	0.865	1.0557	0.350
H_2SO_4 combined	0.1929	0.1307	0.1390	0.083	0.1922	0.2145	0.15	0.158	0.1423	0.132
Ratio $\frac{H_2SO_4\ p}{H_2SO_4\ c}$	8.88	12.28	9.06	12.6	10.8	13.3	10.8	5.5	7.4	2.65
Total N	9.358	7.670	10.51	5.69	12.57	15.42	10.93	5.73	6.627	4.6
N of NH_3 grms.	0.5509	0.4184	0.4887	0.46	0.5025	0.5672	0.3986	0.29	0.3818	0.196
N of NH_3 (per ct. of total N)	5.9	5.5	4.1	8.1	4	3.6	3.6	5	5.8	4.3

	Case VI.				Case VII.		Case VIII.		Case IX.	Case X.
	Jan. 26, 1900.	Jan. 31, 1900.	Feb. 21, 1901,	Mar. 4, 1901.	Jan. 10, 1900.	Apr. 30, 1900.	Feb. 11, 1902.	Jan 17, 1903.		
Volume	600	570	700	530	2095	1815	1120	1260	1335	1033
Specific gravity	1023	1019	1020	1018	1012	1010	.....	1011	1016	1018
Reaction	alkaline	acid	acid	sl'ghly acid	alkaline	acid	sl'ghly acid	acid	sl'ghly acid	sl'ghly acid
Albumin	0	0	0	0	0	0	present	large am'nt	0	0
Indican	0	0	slight	very slight	slight	0	0	0	0	0
Urea	11.55	10.52	17.46	11.33	16.68	10.67	19.80	16.79	16.3	12.29
Uric acid	0.3445	0.221	0.272	0.205	0.3163	0.2841	0.1972	0.7210	0.428	0.40
Ratio $\frac{\text{urea}}{\text{uric acid}}$	34	48	64	55	53	37	100	23	38	31
H_2SO_4 preformed	0.8847	0.676	1.20	0.731	1.498	1.113	1.4476	1.199	1.26	0.98
H_2SO_4 combined	0.1146	0.127	0.224	0.192	0.2681	0.1158	0.1884	0.1935	0.316	0.193
Ratio $\frac{H_2SO_4\ p}{H_2SO_4\ c}$	7.7	5.3	5.4	3.8	5.6	9.6	7.69	6.2	4.0	5.1
Total N	5.4	4.92	8.15	5.30	7.771	4.985	9.251	7.832	7.73	5.74
N of NH_3 grms.	0.3967	0.155	0.266	0.15	0.3287	0.3632	0.7920	3.3716	6.35	0.66
N of NH_3 (per ct. of total N)	7.4	3.2	3.3	2.8	4.2	7.2	8.6	4.8	8.2	11.5

TABLE IV.—Continued.

	Case XI.	Case XII.	Case XIII.	Case XIV.	Case XV.	Case XVI.	Case XVII.	Case XVIII.	Case XIX.
Volume	650	840	950	950	1035	465	1500	1375	1400
Specific gravity	1018	1021	1014	1021	1014	1029	2018	1011	1008
Reaction	acid	acid	acid	acid	acid	alkaline	str'ngly acid	acid	slightly acid
Albumin	slight	0	0	0	0	0	0	0	faint trace
Indican	moderate	0	0	0	0	0	faint trace		
Urea	14.47	16.28	18.94	22.53	9.396	14.35	20.78	12.71	9.91
Uric acid	0.428	0.3326	0.2971	0.4639	0.2851	0.54	1.706	0.066	0.141
Ratio $\frac{\text{urea}}{\text{uric acid}}$	35	49	64	48	33	26.5	12	193	70
H_2SO_4 preformed	1.13	1.4175	1.602	1.6272	0.5555	0.849	1.4993	1.02	0.643
H_2SO_4 combined	0.166	0.2105	0.1470	0.1918	0.1584	0.170	0.2397	0.143	0.134
Ratio $\frac{H_2SO_4\ p}{H_2SO_4\ c}$	6.8	6.7	10.9	8.5	3.5	4.98	6.26	7.12	4.8
Total N	6.76	7.602	8.843	10.53	4.384	6.71	9.709	5.94	4.63
N of NH_3 grms.	0.35	0.5687	0.6824	6.6184	0.1625	0.235	0.7025	0.14	0.28
N of NH_3 (per ct. of total N)	5.2	7.5	7.7	5.9	3.7	3.5	7.2	2.28	6

In Case I. the aromatic sulphates were not increased, but indican was present in every specimen examined (Table V.):

TABLE V.

	Case I.								
	Oct. 26, 1899	Oct. 27, 1899	Oct. 28, 1899	Jan. 20, 1900	Feb. 14, 1900	April 4, 1900	May 14, 1900	May 14, 1901	May 16, 1901
Volume (c.c.)	1130	810	1175	1000	1000	1150	1850	1140	1575
Specific gravity	1023	1027	1024	1023	1029	1023	1016	1024	1019
Reaction	acid	acid	acid	acid	acid	acid	acid	acid	str'ngly acid
Albumin	0	0	0	0	0	0	0	0	0
Indican	moderate	moderate	moderate	strong	present	moderate	moderate	fairly strong	large am'nt
Urea	22.88	20.01	24.13	21.95	26.87	25.6	20.0	30.95	29.86
Urea acid	0.634	0.4512	5.541	0.505	0.648	0.688	0.850	9.932	0.8974
Ratio $\frac{\text{urea}}{\text{uric acid}}$	36.09	44.32	44.6	43.5	41.5	37.2	35.3	33.2	33.0
H_2SO_4 preformed	2.079	1.726	1.978	1.66	2.06	1.942	2.07	2.61	2.2743
H_2SO_4 combined	0.162	0.125	0.189	0.148	0.21	0.180	0.226	0.204	0.2622
Ratio $\frac{H_2SO_4\ p}{H_2SO_4\ c}$	12.8	13.8	10.4	11.2	9.8	10.8	9.16	12.8	8.68
Total N	10.69	9.35	11.27	10.25	12.56	11.96	14.02	14.46	13.95
N of NH_3 grms.	0.588	0.468	0.650	0.68	0.49	0.49	0.54	0.63	0.8158
N of NH_3 per ct. of total N	5.5	5.0	5.77	6.6	3.9	4.1	3.97	4.3	5.8

The amount of uric acid excreted showed no uniformity in the different specimens. In some cases uric acid was in excess, in others it was in less amount than normal. The average of all the specimens showed a ratio of uric acid to urea of 1:50, or a normal ratio. (See Tables IV. and V.)

In the graver forms of acid intoxication, as in diabetes and certain forms of mania, the acid excretion is in so large quantities that there is a marked increase in the ammonia excretion to neutralize this acid. In the cases of rheumatoid arthritis, however, the percentage of ammonia is not increased, so as to be an indication of a condition of acidosis. The normal percentage of the nitrogen of ammonia is from 2 to 5 per cent. of the total nitrogen. In only twelve of the specimens examined above does it exceed 6 per cent., the average being 5.4. (See Tables IV. and V.)

In reviewing the above records of patients with rheumatoid arthritis, it is found that in all cases with progressive lesions there are certain definite evidences in the urine of perverted metabolism. In only three cases were the stomach contents examined, and in all of these there was absence of free hydrochloric acid after a test meal, although the patients had no other symptoms of gastric indigestion. Intestinal putrefaction was found to be constantly excessive in those patients who had active symptoms of the disease. Besides these evidences of gastric and intestinal disorder there was regularly found in the urine organic acid other than that normally present. As the various forms of organic acid are readily oxidized in the body, the amount formed is probably in excess of the amount excreted.

Patients leading as inactive lives as those with rheumatoid arthritis would naturally be subject to gastrointestinal indigestion, but the regularity with which such symptoms are noted and their degree would suggest that certain of the disturbances of nutrition which occur in rheumatoid arthritis are due to these putrefactive processes in the intestine.

In conclusion, I wish to acknowledge my indebtedness to Dr. Herter in the carrying out of this work. The presence of acidosis in rheumatoid arthritis was first noted by him, and his records of cases are included in the above summary. I wish, also, to express my gratitude to those physicians who placed their patients under observation, especially to Dr. V. P. Gibney, Dr. A. Lambert, and Dr. C. A. Ransom.

THE DIAGNOSTIC VALUE OF LEUKOCYTOSIS.[1]

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of Fort Wayne, Ind.

The remarkably large fluctuation in the number of leukocytes circulating in the peripheral blood constitutes one of the most intensely interesting of hæmatological phenomena. The relationship

[1] Read before the Indiana State Medical Society, May, 1904.

of these changes to different morbid processes has been the subject of the most extensive clinical and experimental study, with the view, among other things, of establishing working, diagnostic, and prognostic formulæ. This, indeed, was done, but with more haste than discrimination, and without due regard to the anatomicophysiologic foundation upon which they ought to rest. Growing out of the resulting disappointment, and with a similar disregard of the fundamental facts above referred to, there has been in certain directions a distinct tendency to greatly depreciate or even reject the phenomena of leukocytosis as a substantial clinical aid. It must be admitted that the variations in the number of leukocytes show some extraordinarily bizarre tendencies which make it difficult or even impossible to formulate rigid and inflexible rules. This, however, is also true of the temperature and pulse curves, to which are nevertheless assigned a high value. A careful study of the phenomena of leukocytosis in its physiological and pathological relations will, I am fully convinced, lead to a similar conclusion with reference to the legitimate significance and value of variations in the leukocyte count.

While the purpose of this article is a purely clinical one with special reference to diagnosis, it seems advisable, for the reasons above given, to enter into a preliminary discussion necessarily in the barest outline of the data upon which leukocytic diagnosis rests.

Morphologically there can be recognized several different kinds of leukocytes, distinguished by marked characteristics, such as size of cell, size and shape of nucleus, and presence or absence of granules in the cytoplasm, and the tinctorial reactions of the different elements which go to make up the cell, especially when subjected to the action of aniline dyes.

For the purposes of this discussion the different forms of leukocytes may be briefly summarized as follows: First, the predominant type of cell known as the polynuclear neutrophile, which may be described as a spherical mass of protoplasm 7 to 12μ in diameter, the cytoplasm containing a greater or less number of rather small granules which are neither exclusively acidophile nor basophile, and are, therefore, called neutrophile; the nucleus is characterized by a peculiarly irregular form frequently made up of broken masses connected by fine filaments, the latter often being severed, thus resulting in several nuclei. Second, in numerical importance, the small lymphocyte, a cell from 6 to 9μ in diameter, the cytoplasm free from granules and containing a small round nucleus which nearly fills the entire cell. Third, the large lymphocyte, 10 to 15μ in diameter, with granules, not demonstrable by triacid and ordinary stains, but by the Nocht-Romansky method, and containing a single round or ovoid nucleus relatively small in comparison with the cell, and not staining deeply. Closely related to these morphologically are cells precisely like them excepting that

the nucleus is indented, and which are, therefore, called transitional forms, and the large mononuclear leukocyte. Fourth, the eosinophile cell, precisely like the polynuclear neutrophiles in every respect excepting that the granules in the cytoplasm are very much larger and have marked affinity for acid dyes, being, therefore, called acidophile, oxyphile, or eosinophile. Fifth, mast-cells varying greatly in size from 7 to 22μ, containing coarse granules which do not stain with either acid or neutral dyes, but with a special technique by basic dyes, hence called basophile. Sixth, and finally, a large cell 10 to 20μ in diameter, occurring in two forms (really two distinct cells), with either neutrophile or acidophile granules, and a single large round or oval nucleus and occurring in the blood only in diseased conditions, but normally present in the bone-marrow, the so-called myelocyte.

These different forms of cells are present in health in somewhat varying proportions, about as follows: polynuclear neutrophiles, 60 to 75 per cent.; small lymphocytes, 20 to 30 per cent.; large lymphocytes and transitional forms, 4 to 8 per cent.; eosinophiles, 0.5 to 5 per cent.; basophiles usually less than 0.5 per cent.

Concerning the biological character and relation of these different forms of cells, two opposing views are entertained: first, that of the Russian school, following the lead of Uski, which regards the lymphocyte as the youngest cell, and the other forms as representing evolutional changes incident to the age of the cell, in other words, that the neutrophile is an old lymphocyte; second, the view of Ehrlich, that the lymphocytes, neutrophiles, and eosinophiles have an independent origin and a complete autonomy of existence. The latter view is the one most generally accepted, and the only one which the writer believes to be supported by the facts. It is, moreover, vital to a correct interpretation of a differential leukocyte count, which should be made in substantially every case.

In the clinical study of the ordinary forms of leukocytosis, the lymphocytes, neutrophiles, and eosinophiles are the ones which principally concern us, too little being known about the mast-cells and other forms to assign them an important routine clinical role for the present at least. In regard to the three groups of cells above mentioned, everything appears to indicate that they have not only a separate origin with characteristic morphology and staining reactions, but that, as would naturally be expected, their functions are widely dissimilar in character. The neutrophile is the form of cell which is especially increased in the ordinary clinical forms of leukocytosis. By an increase is meant a larger number of these cells in a definite volume of peripheral blood than is normally present, the increase being brought about by various influences, some of which are understood, while others are not. The discovery of the phenomenon of positive and negative chemotaxis furnishes the key to the physiology and the clinical interpretation of this type of leukocytosis.

It is now well established by an overwhelming mass of clinical and experimental evidence that the introduction of certain chemical substances into the blood causes a rapid increase, perhaps usually preceded by a transient decrease in the number of these cells in the peripheral circulation. Among these are the toxins of certain organisms, such as the streptococci and the staphylococci; while others, such as those of the tubercle bacillus, bacillus typhosus, etc., either do not produce such an increase or may possibly have a diametrically opposite effect. In order that these chemotactic influences may be manifested it is necessary for the chemotactic substances to gain an entrance into the circulation—a point which is absolutely vital in the whole question of the diagnostic bearing of leukocytosis, and the neglect of which has led to much confusion. Especially is this true in its bearing upon the absence or presence of suppuration in some part of the body. This particular question is one of the most important and most frequent in this connection, and it is just here that the most conflicting observations have been recorded. In one of my own cases, for instance, an obscure subacute localized pulmonary lesion of some sort existed, the exact nature of which was in doubt, although the history rather pointed toward an abscess. Confirmation of this view was looked for in the leukocyte count, with entirely negative results. In the course of forty-eight hours after the patient entered the hospital the diagnosis was cleared up by the evacuation of an enormous pulmonary abscess into the bronchial tubes, which very nearly suffocated the patient, an excellent recovery following the spontaneous discharge of pus. It is very probable in this case that a thick, impervious, pyogenic, non-absorbing membrane had walled in the pus from the circulation, and had prevented the entrance into the circulation of the bacterial toxins which probably constitute the chemotactic substance in cases of suppuration. This same question must be kept in mind in the diagnosis of suppurative appendicitis, where perhaps more often than elsewhere the question of the existence or non-existence of a pyogenic process presents itself for solution. If the anatomical conditions are such that the toxins cannot to any great extent enter the circulation, a leukocytosis of diagnostic significance may be entirely absent. There is another feature, however, with which the diagnostician always has to deal, and that is the reactive or resistive power of the individual of which the leukocytosis is an expression. We must recognize in the occurrence of an active leukocytosis under such conditions the marshalling of the defensive machinery of the organism against an invading foe. If, now, the organism is overwhelmed by the intensity of the morbid process or from pre-existing disease, and lacks the ability to make an effective resistance, the leukocytosis will not appear. It also seems possible that peculiar idiosyncrasies may occur here as elsewhere.

Keeping in mind these conditions, the general proposition can

be made that the presence or absence of a suppurative process is indicated with considerable certainty by the presence or absence of a neutrophile leukocytosis, if other chemotactic influences can be eliminated; but, of course, like most diagnostic criteria, it must be weighed and checked by all the facts in the case.

The remarkably brief time intervening between the occurrence of the morbid excitation and the development of the leukocytosis precludes the possibility of an increase in the total number of leukocytes at all commensurate with the increase found in the peripheral circulation. The only other hypothesis and the one now generally accepted is that they are in large part stored in a quiescent state in some of the internal channels of the circulation and are suddenly called forth in response to the demand for their services on the part of the organism. It is believed by some that this storage is in the capillaries of the lungs, but Ehrlich does not hesitate to affirm that the bone-marrow is the only place where such a storage can be found.

There appears to be no doubt that the bone-marrow is the exclusive source of the neutrophile, its appearance there being first in the form of a so-called myelocyte or large mononuclear granulated cell, which, perhaps, never wanders outside of the bone-marrow into the general circulation except in morbid conditions and especially, though not exclusively, in myelogenous leukæmia. This view of the life-history of the neutrophile is established by a chain of evidence, clinical and experimental, which may be regarded as entirely conclusive and separates this cell and its progenitor, the neutrophile myelocyte, from all other forms of leukocytes by a sharply defined line of demarcation.

The eosinophile leukocyte, although not ranking next in numerical importance, will be considered here because of its close genetic relationship and morphological similarity to the neutrophile leukocyte. Owing to the resemblance just mentioned the eosinophile has been frequently regarded as a stage in the evolution of the neutrophile, and has been commonly spoken of as its senile form. This view is not in accordance with the facts, and it may be considered as proven that eosinophile leukocytes circulate in the blood as the progeny of the eosinophile myelocytes pre-existing in the bone-marrow. The behavior of the eosinophile under physiological and pathological conditions can only be explained on this hypothesis and presupposes radical specific differences in the nature and function of the two cells. The final proof of its completely independent existence as a separate cell form is furnished by the discovery of mytosis—a regenerative phenomenon—which is entirely incompatible with senescence. Moreover, under conditions which lead to an increase in the number of eosinophile cells in the peripheral circulation, marked changes have been noted (as, for example, by Opie,[1]

[1] THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, March, 1904.

in his recent article) in the bone-marrow, including among other things marked increase of mytosis among the cells above described as being the progenitors of the ordinary eosinophile leukocyte. Although perhaps not conclusively settled, it seems probable that like the neutrophile and unlike the lymphocyte, the eosinophile responds to certain chemotactic agents. Just what these influences are is known only in part, but the clinical association of eosinophilia with various forms of cutaneous disease, with trichina and intestinal parasites, gives to this blood phenomenon a highly important character. It is worth noting here that a parallelism exists between the behavior of the neutrophile and eosinophile leukocytes under chemotactic influences or at least a similarity in the respective vital conditions which produce or permit a large increase in the numbers of these respective cells. I refer to the reactionary power of the organism against invading infection. Opie has shown, for instance, that in guinea-pigs an intense and rapidly fatal infection with trichina causes a rapid diminution in the number of eosinophiles, while a mild or moderate infection with the same parasite leads to a more or less well-marked eosinophilia, thus indicating that the eosinophiles, like the neutrophiles, are rapidly increased, or at least thrown into the circulation from the bone-marrow in response to a demand on the part of the organism for their functional activities whatever they may be. The theory of Neusser, that their increase is incident to a sympathetic irritation, is not now generally accepted, although the facts upon which it is based are of great interest and certainly lend support to his views, or at least to a close relationship of eosinophilia to the sympathetic. Ehrlich believes that among other things the soluble products of broken-down epithelial cells have the power of exciting positive chemotactic influences upon the eosinophiles, leading to their local accumulation around the sites of ulceration and other destructive processes. An extended discussion of these questions is not within the scope of this paper, and I will for the present leave the eosinophile cell, having endeavored to emphasize two important facts: first, that this cell is specifically distinct and separate from all other cells in its genetic, biological, and functional relations; and, second, that experimental and clinical observation has shown that it responds in a peculiar manner to the influences of certain morbid agents.

With regard to the lymphocytes, their nature and origin are entirely different from that of the neutrophiles and eosinophiles. All the available evidence, experimental and clinical, points to the lymphatic apparatus as the source of these cells. It would be impossible to canvass this question fully within the scope of this paper, and only a few points can be mentioned. Clinically, an increase in their number is very constantly associated with disease of the lymphatic system, and the well-known tendency of tuberculosis and syphilis to implicate the lymphatic glands is the probable explanation of

the frequent occurrence of lymphocytosis in these two diseases. Possibly, also, in pertussis, another disease in which lymphocytosis is frequently noted, the explanation is found in an acute inflammatory swelling of the tracheobronchial lymphatic glands. It has further been observed that the blood which emerges from a lymphatic gland has more lymphocytes in it than when it entered—a fact which is only susceptible of explanation upon the ground of the formation of lymphocytes within the gland.

An effort has recently been made in several quarters to break down the sharp distinction heretofore supposed to exist between lymphatic and myelogenous leukæmia, and incidentally between neutrophiles and lymphocytes, especially because of the fact that in some of the cases of lymphatic leukæmia changes in the bone-marrow have been found. These facts do not appeaar to me to justify so sweeping a conclusion, and, indeed, they are not at all new, as Ehrlich a number of years ago recorded changes in the bone-marrow in lymphatic leukæmia and reported at least one case in which the bone-marrow was almost entirely converted into lymphoid tissue. Among the pathological facts pointing to a very sharp distinction between the nature and function of lymphocytes and neutrophiles may be mentioned the observations of Milroy and Malcolm, who found an increase of alloxur bodies in the urine in myelogenous leukæmia, but found no such increase in the lymphatic form. While much remains to be learned before the subject can be fully cleared up, it appears justifiable at present to regard the occurrence of a lymphocytosis as the clinical evidence of irritative lesions of the lymphatic system.

Keeping in mind, now, the biological facts above set forth in brief outline, and omitting for the sake of conciseness and clearness of conception of the main facts, certain subordinate or obscure points, such as the mast cells, the transitional forms, and the broad distinctions between the large and small lymphocytes indicated by the Nocht-Romansky method, we are ready to ask ourselves for more specific answers to the various questions concerning the significance of leukocytosis in general and the methods and precautions necessary for its proper interpretation. In a general way it may be said that there are two broad types of leukocytosis: one due to an irritative lesion of the lymphatic apparatus represented by a lymphocytosis, the other due to chemotactic influences and represented by a neutrophile or eosinophile leukocytosis, according to the specific form of chemotactic influences at work. This being the case, it is obviously necessary that not only the total number of leukocytes should be known, but the relative number of these three predominant types. This can only be determined by the dried specimen in the form of a thin film on cover-glass or slide properly fixed and stained in such a manner as to differentiate these various forms of leukocytes and determine the proportions of each present in a con-

siderable number of stained leukocytes, say, for instance, 500. For this purpose Ehrlich's triacid mixture, followed by methylene blue to accentuate the nuclei, is the best staining method, as it makes possible the differentiation of the different forms of granules leading to the recognition, among other things, of the myelocytes when present in the blood. The ordinary eosin hæmatoxylin method, which is more easily applied, enables us to recognize the three types of cells already referred to, with certain limitations of error perhaps too small to affect conclusions from a clinical point of view. By this latter method the neutrophile is recognized by the shape of its nucleus and the absence of visible granules, the eosinophile by its large, deeply stained granules, and its nucleus, which corresponds closely to that of the neutrophile, and the lymphocyte by its size, shape of nucleus, and absence of granules. With Ehrlich's stain, however, the neutrophile granules are distinctly visible, and this stain, supplemented when necessary by a special method for the mast-cells and the Nocht-Romansky method above referred to, is in every way to be preferred.

The essential point, however, is that in connection with every leukocyte count there should be a differential count of a properly fixed and stained specimen, with a view of determining the proportional number of the different forms present. In some cases it will be found that the increase is due almost entirely to lymphocytes, while in another it is the neutrophiles or the eosinophiles, or the increase may affect several types of cells at one and the same time, though it is nearly always the case that one or the other form of cell will be sufficiently increased to enable one to label the leukocytosis as belonging to a particular type.

In attempting to make an analytical study of my own experience I collated some seven hundred counts in a large variety of diseases, mostly of a chronic nutritional type, but find the task of their detailed study altogether too large for the scope of this paper, and have, therefore, selected 131 consecutive cases of chronic gastritis, with a view of studying the general character of these cases and determining what light, if any, the leukocyte count would throw upon the general features of a case. In substantially every case upon which the classification is based the leukocyte count was made during the progress of digestion. Furthermore, the cases are divided into six groups, the first comprising those of a leukocyte count with 1000 to 3000; the second, 3000 to 5000; the third, 5000 to 10,000; the fourth, 10,000 to 15,000; the fifth, 15,000 to 20,000, and the sixth, over 20,000. The method sometimes adopted of giving the average count in a particular type of disease is obviously absurd, and can lead to no rational conception of the meaning of leukocytosis and its character in that disease. As illustrating the force of this remark, I recall the report of a series of cases of pneumonia in which by the fallacy of averages, if the individual counts

were not examined, it would have been made to appear that the count in the cases which recovered was the same as in the fatal cases. In the fatal cases the count ranged from a very low count of perhaps three or four thousand in the cases which were overwhelmed by the infection and failed to offer any resistance to those of a very high count of twenty-five or thirty thousand, and it so happened that when these individual counts were added together and divided by the number of cases the result was almost precisely the same as the average in the milder cases.

It is only by dividing a considerable number of cases of particular diseases into groups based on the leukocyte count and studying these groups separately that any practical conclusions of value can be reached, or at least a rational attempt made to throw some light upon the question as to whether the number of leukocytes present in the peripheral blood has an important or substantial bearing upon the main clinical questions with which we are usually concerned.

So far as this particular series of cases is concerned the following tabular statement gives the result of such an analysis:

LEUKOCYTES IN 131 CONSECUTIVE CASES OF CHRONIC GASTRITIS.

Leukocytes	1000 to 3000	3000 to 5000	5000 to 10,000	10,000 to 15,000	15,000 to 20,000	Over 20,000
Cases	10	17	70	27	5	2
Per cent.	7	13	53	21	4	1½

In considering the leukocyte count in these cases it should be stated that in a large number of them, especially the earlier cases, no differential count was made. The only inferences, therefore, that can be drawn are as to the total number of leukocytes, considering the number of leukocytes to be of normal proportions so far as the different types are concerned. While this lessens their value and is a defect which should be remedied in all further work along these lines, yet it is undoubtedly true that a very large proportion of all cases of leukocytosis is of the type just indicated, or, rather, that the increase affects the neutrophiles more than any other form of cell. Any conclusion, therefore, that can be based upon such a study would not be greatly invalidated.

In carefully studying the groups of cases of gastritis made with reference to the leukocyte count, I have been unable in the first three groups ranging from one to ten thousand to discover any striking characteristics belonging to each one. Their response to therapeutic measures and the general severity of the morbid process were very much the same in one group as in that of another, excepting that in the first group in which the leukocyte count ranged from

one to three thousand there appeared to be a somewhat severer grade of general debility and lack of vitality than in the other two. This, however, was not very marked, and the number of cases was somewhat too small to base a definite conclusion upon them in this respect. Moreover, these features of individual cases were more accentuated in some of the patients with higher leukocyte counts than in those with lower. Ehrlich himself, I believe, does not attach great importance to the condition known as leukopenia, and further observations will be required to settle this point.

When it comes, however, to the cases with counts ranging from ten to fifteen thousand there is in a large proportion of them strong evidence of the sort of infection which is commonly designated autointoxication of gastrointestinal origin, and I am of the opinion that leukocyte counts of this range have some diagnostic significance in this direction. With reference to the cases with a count of from fifteen to twenty thousand, this is still more marked. The two cases with counts above twenty thousand were complicated, one with tuberculosis with a mixed infection, and the other with a chronic infection of unknown nature, which proved fatal, no opportunity being given for an adequate study of the case.

There are many other questions concerning leukocytosis which it was my intention to canvass in this paper, but the proportions which it has already assumed make it necessary to close at this point.

The following conclusions appear to me warranted at the present time:

1. A routine enumeration of the white cells in the peripheral blood is of sufficient importance to be made a regular procedure so far as possible in all cases.

2. A single leukocyte count is entirely insufficient as a basis of conclusion in any given case, and should be followed up by several made under different conditions.

3. An increase beyond ten or twelve thousand leukocytes in the peripheral blood indicates varying grades of intoxication, with chemotactic substances of some sort or another.

4. Whether it indicates suppuration or not is a question to be determined by carefully weighing all the facts in each case.

5. The leukocytes indicating suppurative and allied processes are of the neutrophile type.

6. The eosinophile form of leukocytosis indicates among other things, and perhaps principally, cutaneous or parasitical diseases in the intestine or elsewhere.

7. Lymphocytosis clinically signifies an irritative lesion of the lymphatic apparatus.

8. A differential count should be made in all cases to determine the type of cell which has been the subject of the principal increase where such increase exists, and such records carefully kept and

collated as a basis for the determination of the clinical significance of leukocytosis in the future.

9. In the diagnosis of malignant disease a leukocytosis is of very subordinate value, and when present is probably not due to the malignant disease, *per se*, but to coexisting chemotactic toxins.

THE PRESENCE OF AIR IN THE VEINS AS A CAUSE OF DEATH.

By James S. Greene, M.D.,
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Ever since my graduation thesis was published under the above title[1] in 1864 I have thought that some time I might prepare a supplementary paper. The present article is an attempt to put the thought into execution. While scientists and observers have had their periods of productive activity, it seems to be time for the mere chronicler and commentator to take up his task. In attempting this in supplemental form I shall hope to escape the charge of egotism if in the one paper I sometimes refer to the other and quote a passage here and there.

Forty years ago the subject had been slumbering for a quarter of a century. The preceding period of active interest had culminated in the vote of thanks to Amussat at the close of the discussion which followed the report of Bouillaud[2] to the French Academy of Medicine.

The practical result of this long academic dispute was, as well expressed by Couty, "to make the entrance of air a classical question;" all surgeons knew the danger of certain cervicothoracic venous wounds and the number of cases in operations rapidly diminished. Besides the case of Barlow,[3] omitted from my earliest list, a fatal case was reported by Trélat[4], and like accidents with recovery are recorded by Armsby,[5] Cheever,[6] Tadlock,[7] and Courvoisier.[8] At the present day surgeons, without ignoring or belittling, have practically eliminated the danger in operations within the recognized danger space. They proceed though they hear the warning sound, but they put on instant pressure at the proximal end of the wound. Of course they avoid the conditions which may invite trouble.[9]

When I wrote a new-found variety of the accident was coming into view, and I aimed at a re-awakening of interest. Cases of death by the entrance of air through the uterine veins were then little known, and there was again, as in the days before Amussat, much skepticism. Years followed, during which general assent prevailed regarding the actual dangers of the accident; and the

acknowledged danger region, already including the uterine veins, had also come to include the sinuses of the dura mater.[10] History still repeats itself. Within the last ten or fifteen years skepticism again has found expression; but while the earlier doubts had their excuse in paucity of recorded facts, the later ones are in some measure due to the overlaying of facts upon facts, with resulting confusion of inference.

In the earlier paper I gathered notes of 11 cases attributed to spontaneous entrance of air into the uterine veins, and 4 cases of forcible injection of air into the uterus. With these were brought into comparison an analogous class "in which gas seems to be developed within the vessels instead of admitted from without." "In fact," I wrote, "there appears to be a regular gradation of instances from such as are physiological to such as are clearly due to the operation of physical laws." After citing instances I added: "It is impossible not to associate this class of cases with that immediately under discussion; and we must admit the possibility of some peculiar and unfrequent condition of the system which predisposes to the reception of the noxious agent or to the more rapid development of fatal effects." And quoting further: "In those cases where gas is unquestionably developed within the vessels it would be useful to investigate its exact composition, and thus determine whether it is generated by decomposition of the blood or merely set free from solution;" added, "the gas might be collected in an inverted beaker and measured approximately, and in a case admitting of doubt whether the gas was atmospheric air or not the rough analysis resorted to by Roux would suffice." Now it is singular and noteworthy that no chemical analyses of gas found in the heart or vessels have since been recorded with one exception to which I shall refer later.

In 1863 Pasteur,[11] then just elected a member of the French Academy of Sciences, received the unanimous award of its *Prix de Concours* for his "memoir upon the organized corpuscles existing in the atmosphere," a memoir which gave the *coup de grace* to the theory of spontaneous generation. From this epoch-making discovery have developed the stupendous and ever-extending achievements of modern bacteriology.

Now let us leap the intervening forty years and ask what is the present attitude of bacteriology toward the venerable subject of Air in the Veins. Professor Welch[12] tells us that to *gas sepsis* "belong most of the cases which have been reported as deaths due to the entrance of air into the uterine veins." "No case of alleged air embolism from the uterus," he says further, "however plausible, can be considered as positively proven without a satisfactory bacteriological examination."

This opinion is founded on the fact that bacillus aërogenes capsulatus has been discovered in cases of uterine infection where gas

has been found in the tissues and vessels after death. Dr. Welch states that "the limitation of gas to the right heart and adjacent vessels may occur from invasion of the gas bacillus, and is not, as is often represented, peculiar to air embolism." He does not cite any cases illustrative of this statement, and we may accept the fact on his high authority; but it would, nevertheless, be extremely interesting to read the clinical history and the minute post-mortem conditions found in such a case. It must be conceded, as was regretfully conceded forty years ago, that published cases are often sadly inadequate and inconclusive, and we may sympathize with Dr. Welch in the lament that: "Did we not know how long it takes new knowledge . . . . to penetrate throughout the medical world it would be amazing that cases should still continue to be reported . . . of deaths ascribed to air embolism without any bacteriological examination, or even any reference to the possibility of any other explanation."

I cannot forbear to ask, however, if the fervor of the scientist and discoverer has not led him to some overemphasis in statement? I write not from a bacteriological point of view, but, as I have said, only from the historical and clinical viewpoint; but I cannot forget how much the earlier views of bacteriologists have been modified with regard to the virulence of diphtheria bacilli.

The earlier doctrine was that the presence in the throat and nose of the Klebs-Loeffler bacilli was adequate evidence of the presence of the disease in the individual, and of danger of contagion from him; but in April, 1902, a committee of the Massachusetts Association of Boards of Health[13] made a report to that body on Diphtheria Bacilli in Well Persons. I quote from that report this paragraph: "We seem to be justified in concluding, from an examination of the experimental and clinical evidence at hand, that only a small percentage of the morphologically typical diphtheria bacilli found in well persons not recently exposed to the disease are virulent." And again: "They (the virulence tests) bring out the fact that if a healthy person is found to have Klebs-Loeffler bacilli, and there is no connection traceable between that person and cases of diphtheria, the chances are very much in favor of the bacilli being non-virulent."

To-day, according to Solomon,[14] bacteriologists are as far as ever from agreement as to the classification and identification of true and false diphtheria bacilli.

Now may not the bacteriologist hereafter modify his present view regarding the gas bacillus, which is, that its presence in blood-vessels or tissues, even though demonstrated unnumbered days after death, *must* set aside a presumption of air entrance as the cause of death[12] in a case which clinically belongs in that category? And even though he concede nothing, has the bacteriologist the final word? Suppose the pathological chemist should sustain the

obstetrician who makes claim that a given case of sudden death after childbirth is due to the entrance of air into the uterine veins, and should, by analysis of the collected gas, affirm that it was atmospheric air, would not the tables be turned, and would not the burden of proof rest upon the scientist who asserts that the presence of gas bacilli found in tissues after death controverts the theory of air embolism as the cause of death in the case? I believe that bacteriologists know that the gas formed in tissues during life or after death by the agency alike of aërobic and anaërobic bacilli is a compound of hydrogen and carbon dioxide; but I do not think that they have published this knowledge to the world. They also suppose that this gas mixture or compound is a result of fermentation of sugar formed by decomposition of proteids or carbohydrates in the system, or of sugar already present in the blood (in case of diabetics), through the agency of some one or more varieties of gas-producing bacilli.

In the mean time there is the fact recorded by Jürgensen,[9] that Hüfner analyzed the gas collected from abscesses behind sterno-clavicular articulation and from the upper part of the thigh, and found nitrogen, 84.45 parts; oxygen, 14.05 parts; carbonic and sulphurous gases, 1.05 parts; but whether the gas was developed in the abscesses or found its way thither from without, both in this case and in that of an analysis by Dressler, there was doubt.

It is nearly ten years since the discovery of the bacillus aërogenes capsulatus, the agency of which is invoked to discredit the great mass of evidence accumulated in medical literature showing the danger of air entrance through the uterine veins, and is it "anything less than amazing" that we have to go back to the time of Roux[15] in the early years of the last century to find more than one recorded statement of measurement or analysis of gas found post-mortem in the heart or vessels?

Now let us examine the single observation to which I have alluded. It is not the observation of a bacteriologist. Indeed, it was published when bacteriology was barely emerging from its chrysalis state. Moreover, it is an observation which supports neither the doctrine of the demonstrators of gas sepsis, nor that of the advocates of air embolism, but illustrates that conjectural *terra incognita* or possible middle ground between the territories, equally definite, as I must maintain, of gas sepsis due to bacilli and of air embolism. It seems an instance fairly demonstrated of gas set free from solution in the blood. The case is recorded by M. Hervieux.[16] A young woman was delivered of her second child, a male, after normal labor. On the tenth day a vaginal injection was administered for the correction of fetid lochia. Careful precautions were taken against the admission of air. The eleventh day another injection was given, with the same precautions. This second injection at 7.30 P.M. was followed by the loss of 750 grams of fluid

blood, and by a rigor and chattering of teeth. Later in the evening she had an angry quarrel, ending with an ungovernable nervous crisis. Death half an hour after midnight.

Autopsy was made thirty hours after death. There was no evidence of putrefaction. Cardiac vessels were carefully tied. Heart distended with gas; vena cava the same; contents of latter lost by accidental puncture, but whole inferior cava from right auricle to primary iliacs full of frothy blood. None in iliacs nor utero-ovarian veins, nor superior cava nor its tributaries. Uterus showed no vestige of inflammation nor suppuration. Washing of the interior at fundus revealed the seat of the hemorrhage: two little erosions the size of a pinhead, with small red clots adhering. Appendages perfectly healthy. Gas from heart, chiefly from right cavity, but some from left, collected under water to a volume equalling 40 to 45 grams of water, analyzed with the following result: oxygen, 7 parts; carbonic acid, 11 parts; nitrogen, 82 parts. Same gases as those of air, but in different proportions.

M. Hervieux cites M. Durand-Fardel's case reported at the Academy of Medicine, session of December 9, 1851. A lady aged fifty-six years, of healthy appearance, in coming out of a bath felt oppressed, sank in a chair without power to speak, and died in five minutes. At the autopsy, twenty-two hours after death, the heart was found to be much distended and to contain bubbles of gas, with very frothy blood. The whole abdominal venous system was engorged with dark, frothy blood, and the blood of the splenic and portal veins contained numerous bubbles of gas.

M. Durand-Fardel adopted the view of a spontaneous exhalation of gas during life in the venous system, probably due to an unknown alteration of the blood.

The analogue of these and similar observations is found in the caisson illness. It seems to be determined that symptoms and lesions in that disease are due to the presence in the system of gas withdrawn from solution in the blood.[17]

Hervieux's case was not one which would to-day be claimed as one of air embolism, but rather as an instance of gas sepsis. Its long interval of ten days after delivery, and its interrupted progress toward the fatal issue would remove it from the former category and align it with the latter. The advocates of gas sepsis might claim it as their own but for the counterevidence of the gas analysis.

The puerperal cases on which Professor Welch relies for the support of his contention are all cases of emphysema of uterine walls, and in all but one there was physometra. Such cases develop slowly, either days after delivery or incidentally to the presence in the uterus of a decomposing fœtus. They form clinically a class by themselves. The reasonable inference in these cases is that the bacillus aërogenes capsulatus found *post-mortem* in the tissues is the fatally noxious agent; but the weak point in the demonstra-

tion is the omission to collect and analyze the gas found in the vessels. When death has occurred suddenly, after hours or days of localized emphysema, the event may well be suspected to be due to distention of the right heart or invasion of the pulmonary capillaries by the gas after long overdistention of neighboring or remoter tissues.

Cases of injury or operation with complications such as gangrene and emphysema are recorded in which slow or sudden death has ensued, and the veins and heart after death have been found to contain frothy blood.

They form a class wherein the presumption now is that the gas found post-mortem is the product of sepsis. Whether the presence of this gas in the heart and pulmonary vessels be the proximate cause of death is a question which should be decided in each instance by the clinical history and the quantity and distribution of the gas.

Many cases of this character were recorded before the discovery of gas bacilli, and attributed by the observer to slow or rapid admission of atmospheric air.[18] Of these only one (referred to by Dr. Porter) is found in surgical history of modern war.[19]

It is curious to note that notwithstanding Baron Larrey's[20] well-known remark concerning the frequency of deaths on the battle-field from entrance of air into the veins, neither the index of the "Surgical History of the War of the Rebellion" nor that of the Chino-Japanese War contain any reference to air embolism.

Parise[21] affirms that the putrid character of the gas developed in some cases of gangrene determines no septic action, but produces death in the same way as does air accidentally entering the veins.

Professor Welch himself admits the "possibility that gas generated by bacteria may have entered wounded veins in sufficient amount and so suddenly as to have caused death."

The surprising thing is that this theory of gas sepsis should be invoked to invalidate a well-established presumption of death by air entrance in a class of cases wholly unlike these septic cases in their clinical aspect, and this without a single supporting verification by gas analysis. On the other hand the clinical picture of spontaneous air entrance through the uterine veins is identical in feature with the same accident occurring in surgical cases and in attempts at abortion. It occurs at the time of delivery or shortly afterward, is sudden, and is seldom lacking in some of the characteristic symptoms.

Take, for instance, the case of Cordwent.[22] A healthy young woman of twenty-eight. Babe and placenta expelled while the patient was standing. Babe fell to the floor, dragging out the placenta. Almost immediately a kind of gurgling sound was heard. Patient remained standing about a minute, holding on to the bedpost, and then exclaimed: "I cannot see; I feel faint; lay me on the bed." This was done, and she died instantly. Autopsy

twenty-four hours after death. Uterus healthy. A portion of wall of fundus about the size of a five-shilling piece, slightly more puffy than the other portions; and on cutting into it air-bubbles escaped. There was no laceration of placental surface. The uterine cavity contained only one small clot; its lining membrane was healthy.

The coronary vein of the stomach was distended by air, and contained only a fine thread of blood. The right side of heart was slightly gorged, and when the auricle was punctured air-bubbles escaped from its contained blood. There can be no mystery about the mechanism of this fatal event. The uterus relaxed after the pain which caused the rapid expulsion of the child, and produced a partial vacuum. The inrushing air at the moment when the placenta passed through the vaginal outlet found entrance into the venous system through uterine sinuses not wholly closed by coagula. The inlet was sealed by collapse of the vaginal walls and perhaps by a clot, and the ascent of the imprisoned air to the heart was favored by the erect position of the patient.

Other cases have been reported by Heckford,[23] Davidson,[24] and Braun.[25] Lauffs[26] gathered notes of 18 cases which he classified as cases of spontaneous air embolism through the veins of the uterus. Nine of them I have in this or the earlier paper cited from the original sources. Two others, reported by Draper,[33] occurred in the offices of abortionists, and may have been forced rather than spontaneous, while three, observed in the clinic and polyclinic of Bonn[18] and elaborately reported, were cases of artificial delivery in which it would be difficult to exclude the possible agency of shock or sepsis or both as contributing to the fatal results, though air was found at the autopsy in the right heart and in the large veins in each case. There remain the four following cases to be credited to Lauffs: Levy[27] reported a case of a woman of fifty years who died while undergoing a delivery by forceps. At the autopsy were found partial loosening of the placenta, frothy blood in abdominal veins, air in right internal spermatic vein and in pulmonary vessels. Fürst[28] reported a fatal case occurring in a Vienna hospital. A woman aged twenty-five years, secundipara, who on expulsion of the infant was turned from side to back. Thereupon she immediately became asphyxiated, lost consciousness, and died in an hour after delivery. The autopsy revealed air embolism as the cause of death.

Bischoff[29] gave a case recorded by Hegar. A woman who had experienced a normal labor died suddenly on the fifth day of lying-in. She had laughed loudly while engaged in conversation, and immediately convulsive movements and rattling in throat ushered in death. Autopsy showed frothy blood in right ventricle and right branch of pulmonary artery. Air escaped from veins of the dura. These three cases, while suggestive of nothing so much as spontaneous air embolism, are too meagre of detail to permit of exact

conclusions. The fourth, which I condense in extracting from the paper of Lauffs, is a well-observed case of Kezmarsky[30] occurring at the University Lying-in Clinic of Budapest, March 19, 1877.

An unmarried washerwoman of thirty years was admitted at 10 A.M. in labor at full term of her fourth infant. Hydramnios was diagnosed. From noon the pains, which had been few and weak, became frequent and stronger. About 3 P.M. a bag of membranes emerged, the size of a fist, and during the momentary absence of the midwife from the bedside, burst, flooding the bed with the waters. About fifteen minutes afterward, while the woman was still clearing up the water, the patient suddenly jerked her head backward, gasped painfully for air, her face became livid, and her breathing stopped. The whole scene passed with such lightning quickness that the assistant, Dr. Liebmann, who reached the bed in two minutes, found no pulse, but noticed two incomplete respiratory movements of the thorax, and heard the heart's action faintly as the patient lay on her back. While artificial respiration was undertaken, Dr. Liebmann applied forceps to the child's head in the pelvis, and delivered an asphyxiated male infant of 2780 grams' weight. External pressure caused the expulsion of the placenta directly afterward. In spite of all efforts the heart's action ceased and the patient expired. The liquor amnii amounted to 4 litres. At first glance Dr. Liebmann had the impression that eclampsia might have occurred, but in contradiction of that idea came the decided assurance of the practitioner present and the experienced midwife, that there were no convulsions. The belief was then adopted that either pulmonary embolism or the entrance of air into the veins was the cause of death. Autopsy twenty hours after death by Professor Scheuthauer. The heart was moderately contracted. The left ventricle contained a little fluid blood of yellowish-white color. In the right ventricle, especially near the pulmonary valves, also in the inferior cava and the uterine veins, was pale reddish-brown fluid blood mingled with small and large air-bubbles. Frothy blood flowed from the veins of the uterus when that organ was incised and pressure applied. Professor Scheuthauer thought that the air present was not the product of putrefaction, because the body presented no signs of decomposition, and there was no trace of air found in the jugulars nor the other large vessels.

Kesmarsky remarks concerning this case that the manner of entrance of air was obvious. At the rupture of the membranes the woman was nearly in the Sims position, for she lay on her left side with thighs drawn up, pelvis oblique over the bed, and head on the edge of two flat hair bolsters, so that the face and thorax seemed a little downward. The prominent abdomen was lower than the pelvis, and noticeably lower than the *ostium vaginæ*. The mechanism of the accident was as follows: When the large bag of membranes was suddenly extruded and ruptured, a space was made

between the collapsed membranes and the flabby, voluminous walls of the multiparous vagina. At the same time the intra-abdominal pressure, by the quick escape of a very large quantity of amniotic fluid, became negative, and air rushed in between the membranes and the walls of the uterine cavity. The fetal head descended and the retracting cervix uteri closed around it. The placenta, having become partially detached by the contracting and relaxing uterus, left openings into uterine sinuses through which the air, imprisoned in the uterus, directly entered the veins. All this occurred within the five minutes which elapsed between the bursting of the membranes and the first symptoms of difficult breathing. The absence of external hemorrhage has no value as opposition testimony, because such blood as issued from the placental sinuses was, like the air, prevented from escape by the presence and pressure, as a tampon, of the fetal head.

As though to furnish more links to the chain of proof, surgical cases are not wanting with post-mortem demonstration of fatal air entrance by way of the uterine veins. Wyman[31] and Cheever[6] have each reported a case occurring during aspiration of pelvic tumors. Several instances of sudden death in cases of placenta prævia have been recorded and discussed in German periodicals, and are reasonably ascribed to air embolism. To this category belong cases of Kramer, Kruckenberg, and Heuck.[32] But when in such cases, those for example of Lesse and Zorn,[32] death has been much delayed, the door of controversy is opened as to the possible agency of gas sepsis. The eight cases which I have chosen for citation, added to the eleven collected in my thesis, all lack, it is true, the ultimate proof of the chemical analysis, while some are extremely meagre; but all the reported cases of supposed gas sepsis are likewise unsupported by chemical proof. If the latter class stands on a bacteriological basis, surely the former may claim an equally firm foundation on clinical grounds and convincing analogies.

I suppose that most practitioners of long experience have witnessed cases of sudden death after childbirth, with attendant symptoms indicating the possible entrance of air into the circulation, but have been unable to verify this inference by means of an autopsy.

It is probable that more fatal cases of air entrance through the uterine veins occur than do like accidents in surgical operations, and that present-day obstetricians are less generally cognizant of this danger than surgeons. There would be a correlation between these two facts, if facts they be.

Skeptics have adduced the fact that the accident has not occurred in Cæsarean section. The reply is that since atmospheric pressure does not become negative, conditions promoting the accident are not afforded in that operation. According to Cohnheim, air can find entrance into the veins whenever atmospheric air has access

to the cavity whose tension is less than that of the air. These conditions may be fulfilled in particular cases in a variety of ways:

Veins and sinuses may be emptied by hemorrhage and remain patulous; violent nervous agitations may induce succussion of abdominal walls so as greatly to extend the area of *l'espace dangereuse;* a contracting and relaxing uterus may act as a receiver and pump; or a sudden turning of the body may cause retreat of the viscera, and suction of air through a relaxed and patulous uterus and vagina. Air once introduced may be hindered from escape through the channel of entrance, and the greater veins may easily be the path of least resistance. None of these conditions are presented in the operation of Cæsarean section.

As the controversy stands to-day, in the light of the solitary gas analysis of Hervieux, it would seem reasonable to maintain that no case of assumed death by gas sepsis even with gas bacilli demonstrated could be declared proven, nor any case of assumed death by air embolism could be declared spurious or disproven without collection of the gas found in the heart or vessels, and the demonstration of a pale-blue hydrogen flame on ignition, or an explosion due to the presence of hydrogen or marsh gas.

True, the recorded cases of spontaneous uterine air entrance are rare. So, too, are those of pulmonary embolism after childbirth; and it may be, as has been affirmed, difficult or impossible to distinguish between these two accidents by the symptoms. Pulmonary embolism, however, is usually consequent upon phlebitis and thrombosis of the thigh, and occurs with these concomitants days after delivery, as gas sepsis does with the surroundings of gangrene or decomposition; while spontaneous air embolism, as has been shown, is attendant upon, or follows shortly after, delivery. All these accidents indubitably sometimes occur, and in the interest of scientific truth what is most needed is the urging upon all observers the careful reporting of cases, and the determination in each case of the exact nature of the accident, whenever possible, by post-mortem investigation carried to its last analysis.

The literature of alleged forcible introduction of air into the veins of the uterus has expanded in forty years from the four cases contained in my thesis to a large number.[33]

To the present inquiry these cases have pertinence, first, because, taken as a whole, the cause of death, when confirmed by the finding of air in the right heart and large veins, does not admit of dispute; second, by reason of the striking similarity in symptoms between them and those cases which are chiefly depended upon as characteristic instances of fatal spontaneous air entrance, whether surgical or obstetrical; and it might be added, third, because they offer, especially when the medico-legal element is introduced by a charge of criminality, the best prospect of determining the minimum quantity of air necessary to produce death when admitted into the

veins of the human body. This brings up the third ground of that skepticism to which I alluded to in my opening paragraph, and I shall refer to it later.

Upon the mechanical conditions concerned in the accident, nothing need be added to the remarks already made. Regarding proximate causes it is otherwise, for the period which this résumé is intended to cover is prolific in careful scientific search. It is impossible within the limits here permitted to do more than glance at the conclusions of a few among the noted men who have made patient study of the subject.

In voluminous literature the decade of the 70's, following the revival of professional interest, was surpassed only by the decade of the 30's; in systematic, scientific study and painstaking analysis it surpassed the 30's or any intervening decade, and the impetus thereby given to productive research has extended through the 80's and 90's to the present.

Doubtless both the interest and the results were enhanced by the introduction and the use of instruments of precision unknown to the former generation of workers, and by means of which conclusions could be made much more exact. Panum,[34] in Germany, and Oré,[35] in France, were among the last of the men of note who made contributions to the doctrine of air embolism before the new era opened.

Oré, in 1863, held that air not only distends the right cavities, but also has a sedative action upon the muscular fibre which determines paralysis of the right ventricle, herein taking similar ground to Busse[36] and Marchal (de Calvi).[37]

This chemical theory of Oré, which had some acceptance in France, was contradicted by the studies of Demarquay,[38] who, with his pupil Meric, concluded that air acts only mechanically, herein following the teaching of Magendie.

The theory of Boerhaave and later of Poiseuille,[39] that the lungs are the seat of circulatory obstruction, sustained by Ericcson and Milne-Edwards, was adopted by Claude Bernard[40] in 1873. Muron and Laborde,[41] working together in 1874, maintained the view that brusque injection causes death by distention of the right heart, while injection more slow determines death by suspension of the cerebral functions favored by the diminution of strength of the cardiac contractions. Kowalewsky and Wyssolsky,[42] of Kasan, in 1874, deduced from experiments on cats and dogs a belief in anæmia of the aortic system by the mechanical hindrance of froth in the right heart. Other essayists in the same decade were Pallas[43] and Esleben.[44]

In 1875 a young knight entered the lists to dispute the old doctrines and to establish a new one.

Louis Couty,[45] working in Vulpian's laboratory, made many experiments on animals, together with careful analysis and com-

parison of the labors and theories of all his predecessors in this field, and of all known cases of the accident. On this basis he developed in his thesis for the doctorate in medicine the theory which until recently has held the more general acceptance.

He denied the theories of cardiac paralysis, both chemical and mechanical; likewise the theories that air kills by reaching the brain or by obstructing the lung capillaries. He affirmed, and claimed to have proved, that the entrance of air produces one sole trouble, fall of tension and circulatory arrest; and that all general consecutive accidents, momentary, grave, or mortal, are the direct consequence of this initial circulatory trouble. This stasis of circulation is due not to paralysis of the cardiac walls, for they contract more energetically, but because they contract upon an elastic fluid (air) and compress it instead of propelling it. Further, the distention of the cavities causes tricuspid insufficiency and venous reflux. The blood current into the pulmonary arteries is checked with resulting cerebral anæmia, followed by bulbar death. M. Couty pursued his argument with such eloquence and elaborated it with such wealth of illustration and thoroughness of analysis as to captivate the reader and silence opposition. His doctrine received the seal of Cohnheim's[46] approval, and held undisputed sway for years.

Heart death was the accepted belief of V. Feltz,[47] 1878; Passet,[48] 1886; Jürgensen,[49] 1887. In this country Dr. Nicholas Senn,[50] in 1885, made a monumental study of the subject. He returns to the belief of some of the earlier writers, and espoused by Dr. John Reid, that the conditions producing death are (using Senn's language), "*a*, mechanical overdistention of the right ventricle of the heart and paralysis in diastole; *b*, asphyxia from obstruction to the pulmonary circulation consequent upon embolism of the pulmonary artery."

He believes, further, that "such air embolism is relieved in a comparatively short time provided the contraction of the right ventricle continues unimpaired for a sufficient length of time to force the air through the pulmonary capillaries into the general circulation."

Dr. H. A. Hare, of Philadelphia, in 1889, on the basis of research among the earlier writers and experiments of his own on dogs, avowed his belief in the harmlessness of air entrance into the veins of human beings during surgical procedures. He thought obstetrical cases presented different conditions and might have possibilities of danger.

To give his position its full advantage, I quote the following remark of Dr. Hare, though I have failed to find the paragraphs in his paper devoted to the alleged proof: "We have proved that the quantity of air entering the veins under such circumstances (surgical accidents) cannot be of any great quantity."

Dr. Hare thinks that "there is no evidence whatever that air may not be taken anywhere that blood can flow." But Poiseuille, 1837, is among those earlier authorities with whom Dr. Hare is familiar, and of him Couty remarks: "Le fait (est) bien prouvé par Poiseuille le sang spumeux necessite pour traverser un resean capillaire une pression plus forte que du sang pur." (Thèse, p. 35.)

Velpeau did not attack the proposition that the entrance of air into the veins had been the cause of fatal accidents, as Dr. Hare supposes, but took the true scientific attitude toward the subject, and by analysis of the reported cases, including one of his own, pointed out those which he classed as doubtful, others as probable, and still others as almost certain, not carrying any doubt to his mind. Had Dr. Hare in this spirit examined the record of some cases since Velpeau's time—Volkmann's case, surgery of the head; Trélat's, submaxillary; or Cheever's pelvic case—he must have modified, might have reversed, his opinion.

After these men of the 80's the pendulum of opinion begins to swing away from heart death, though so recently as 1902 we have an American writer, Dr. Malcolm Goodridge,[52] whose research and experiments lead him to the belief that death is due to one of two possible causes:

1. Gaseous distention of right auricle and failure of the blood to reach the left ventricle, and consequently anæmia of the brain centres.

2. And to his mind the more important factor, plugging of the coronary vessels with air emboli.

Hauer,[53] in 1890, holds to the theory of lung death, and with him stands the latest investigator, Dr. L. Wolffs,[54] of Berlin, in 1903.

Dr. Wolffs undertook a series of experiments on dogs to determine what becomes of air injected into the circulation, and what is the cause of death; and his conclusions are noteworthy. He finds that the air entering the venous system presses toward the heart. From the heart the pulmonary arteries are filled with air, which passes on into the capillaries. Here its progress meets with a very important resistance. The heart can overcome this resistance if too great a number of capillaries are not too suddenly obstructed. The heart will meet the increased demand the more easily the greater its strength and the less the amount of blood which has been lost.

On account of the peculiar anatomical relations which exist for the lung capillaries, the air is not pressed into the corresponding capillary system, but into the alveoli, where it mixes with the inspired air of the breath. It is not strange that in this process of filtration isolated air-bubbles find their way into the venous capillaries of the lung. This would again be a cause of death through embolizing of vital tracts in the brain, spinal marrow, or coronary arteries, an outcome surely rare, but one that could not be characterized as lung death.

What becomes of the large quantities of air injected, since it does not enter the general circulation nor remain long in the right heart and large veins, except in cases where the whole pulmonary tract is embolized by too large and sudden injections? Even subtracting absorbed oxygen the remaining amount is still far below the injected quantity. This can only be passed out through the lungs. If the explanation be not accepted that embolized pulmonary capillaries are the seat of interruption of the circulation, how could be explained the greater tolerance in cases of interrupted and renewed introduction of air?

Dr. Wolffs has never been able to find air in appreciable quantities beyond the pulmonary capillaries, neither in the left heart nor in the pulmonary veins, and thinks that only an open *foramen ovale* would permit the passage of air into the great circulation after venous injection. Recorded instances of such transmission he believes to be mistakes in observation due either to decomposition, gas formation *in vivo*, or entrance of air during section.

In all this maze of experiment and inference, always incomplete and often contradictory, is there any clue by which to arrive in sight of a goal of final agreement? Perhaps the clue may be got by twisting a thread from Couty with one from Wolffs. Couty was right when he affirmed the stasis of the blood current, not primarily by overdistention and paralysis of the right heart, but by ineffectual contraction upon an elastic medium and venous reflux through tricuspid insufficiency.

While he admitted in obstruction of pulmonary vessels a possible contributing influence making for stasis, he did not assign to this a sufficiently important role because experiment and deduction combined to mislead him into the belief that, since he found little air *post-mortem* in pulmonary arteries after venous injection, therefore little had entered those vessels. Wolffs having shown that air forced into the pulmonary capillaries does not, as Senn thought, enter the general circulation, but escapes into the alveoli, seems to have supplied, if I may stretch the metaphor, the missing link which connects the threads. The clue is stronger by the two threads than by either alone, and guides to a conclusion. Cerebral and bulbar anæmia is the sole physiological cause of the issue in fatal cases of entrance of air into the veins. The mechanical origin of this anæmia is combined lung and heart stasis in proportions varying with the quantity of air introduced and the time occupied in its introduction. The statement must be thus qualified in consequence of the fact, noted by Cohnheim and interpreted by Wolffs, that, to produce lung independently of heart death, more than half the pulmonary capillaries must be obstructed with air emboli, and that this obstruction must be brusque and not gradual. On the other hand, heart death, unaided, would not occur, because the heart must succumb sooner and by a lower degree of distention

in consequence of simultaneous hindrance to blood propulsion from pulmonary air emboli

There can remain to be noted only those rare exceptions to the foregoing statement, fatalities which are neither cases of lung nor heart death, and which can be explained only by the exceptional passage of air into the greater circulation by way of the pulmonary veins.

In my earlier paper I wrote: "The proximate cause of death has been a subject of much controversy, and it has not yet been explained beyond a doubt;" and after giving a summary of the various views of the authorities then known, I added: "The weight of evidence indicates that one of the chief troubles is the impediment offered to the passage of blood through the lungs." It does not seem that forty years have furnished occasion for much change of emphasis here.

The question already alluded to, of the quantity of air necessary to produce death when admitted into the veins of a human being, is an undetermined one, but must not be disregarded.

I know of no recorded case in which the air found *post-mortem* was measured except that which I have reported above, of Hervieux. In his case the air, collected chiefly from the distended right cavities of the heart, but some of it from the left, equalled in volume from 40 to 45 grams of water. This is definite. A woman died, not instantly but somewhat slowly *post-partum*, and a gas chemically resembling atmospheric air, of a volume equalling 35 to 40 c.c., distending the right heart, was the only cause of death discoverable at autopsy. It does not concern us here whether the gas entered the veins spontaneously or was forced in; whether it was a result of sepsis, or was merely liberated from solution in the blood. We need not too carefully inquire why the state of the pulmonary arteries was not reported upon, nor how a small amount of the gas found its way to the left heart. What we are not permitted to doubt is that the heart was stopped in diastole, and that the distended cavities contained 35 to 40 c.c. of gas twenty-two hours after death. If the pulmonary arteries were likewise distended and their capillaries filled with air or gas emboli, while the ascending cava was charged with frothy blood, such facts would increase the estimated total of gas contained in the circulatory organs at death perhaps threefold or fourfold, and would show that the admission of air or evolution of gas was gradual rather than sudden, and that other mechanical and vital factors, besides cardiac paralysis, might have contributed to determine the character of the symptoms and to promote the fatal issue.

Laborde and Muron found that air injected quickly into the veins of animals caused sudden stoppage of the heart, death by cardiac paralysis. Slow injections by small and repeated amounts the animal supported admirably. Neither slow nor quick, but

gradual, brought lung and brain symptoms before the heart stopped beating.

I give below in parallel columns for comparison the conclusions of a few of the later experimenters, American and foreign, respecting the quantity of air which they found capable of killing dogs. These results vary, but as a whole do not differ materially from those of the investigators who were quoted in my thesis:

ORÉ.	MURON and LABORDE.
60 or 80 or 100 c.c., fatal result.	100 to 200 c.c., *brusque*, instant death.
COUTY.	GOODRIDGE.
80 c.c., *brusque* death; 60 c.c. in weakened dog, death.	75 c.c. for a dog of forty pounds' weight, usually a fatal amount.
SENN.	KOWALEWSKY and WYSSOLSKY.
Dogs weighing about thirty pounds would usually recover after an injection of 30 c.c.; double that amount was generally a fatal dose.	14 c.c. or less at once had killed; 200 c.c. or more in small quantities at a time, borne without disadvantage.
JÜRGENSEN.	WOLFFS.
Slow injection of small and repeated quantities during sixty-five minutes upon a dog weighing 16 kilos, death after 500 c.c.	20 c.c. followed in fifteen minutes by 80 c.c., fatal; 20, 25, and 30 c.c. at intervals covering forty-five minutes, death after 200 c.c. had entered.

I am ready to admit that the fears I formerly expressed regarding the possible mechanical harm from a leaky hypodermic syringe, or from the ordinary operation of tracheotomy, were exaggerated. I do not believe that a bubble of air introduced into a vein is likely to cause alarming symptoms. What may be the minimum quantity capable of doing this in an enfeebled, perhaps exsanguinated, possibly frightened or agitated subject cannot now be affirmed.

Conjectures derived from algebraic formulæ founded on weight and volume comparison in experiments on animals must be delusive.

A statement of Couty is illuminating. After remarking the evidence of cerebral anæmia in animals as only manifest in hurried respiration in the series of observations (the second of his four) illustrating the slightest manifestation of general, and not merely local, trouble, he says: "With man, whose brain is more sensitive, this circulatory trouble, this momentary fall of tension, suffices to produce *a syncope, a momentary loss of consciousness.*" (Italics are Couty's.)

And again he says, after noting the comparative tolerance of some animals: "These accidents are much more rapid with man in whom the heart, we have seen, is more dilatable, in whom the organs are much more dependent, and the brain more susceptible. But even in this more complex organism after the entrance of air the arrest of the central co-ordinators, heart and brain, is not instantaneous; convulsions when they have occurred have super-

vened an appreciable time after the hissing sound—that is to say, twenty or thirty seconds."

The physician cannot overlook the lesson of the larger therapeusis which is constantly enforcing attention to the practical truth that the delicate, nervous equilibrium and the sensitive brain, which accompany the more complex development of the human being, constantly have to be heeded in practice, and must necessarily detach man from comparison with animals, even the superior ones, when there is question of anything in the nature of depressing influence or shock.

It remains but to consider briefly the therapeutics of the subject.

Couty advocated two measures, the one mechanical—bleeding; the other chemical—inhalation of oxygen.

Senn urges, besides indirect treatment by venesection and cardiac stimulants, direct treatment by (*a*) puncture and aspiration of the right ventricle; (*b*) catheterization and aspiration of the right auricle.

These measures he believes to be justified by experimentation as a last resort in all cases where the indirect treatment has proved inadequate to meet the urgent indications. Lauffs, two years later, also suggests puncture of the right ventricle.

Goodridge's experiments were undertaken to determine the therapeutic value of aspiration of the right ventricle, according to the proposal of Senn, combined with venous transfusion of normal salt solution. He concluded that good results may be expected from the combined method of treatment.

The radical direct measures proposed, however well founded in theory and well supported by experiment, are as yet untried in any case of the accident in man.

On the obstetrical side suggestions for treatment are meagre. Some of the more recent books on obstetrics ignore the whole subject; others touch it doubtingly. One of the latest gives reasonable space, but has no word on treatment.

The following remarks are found in the *American Text-book of Obstetrics*: "Very little can be done for the patient even when assistance is close at hand. The cervix should be cleared of clots. Artificial respiration and hypodermic administration of stimulants should be promptly employed. Tracheotomy and the inhalation of oxygen gas, in order to inflate the lungs and to expel the air emboli, have been suggested."

If this utterance be accepted as the concise expression of present-day expert opinion in obstetrics, it follows that the emphasis must be upon preventive measures. These consist wholly in carefully guarding against the risks which recorded experience and tested theory have signalized. It means watchfulness to avoid any conditions which may produce, ever so transiently, a negative atmospheric pressure within the abdomen at the time of expulsion of the contents of the uterus, and for a brief but undetermined period

following. Especially is there need of this if the delivery be rapid, if it be attended or preceded by considerable hemorrhage, if there be placenta prævia, or any need of operative procedures. Care should be taken to follow down the contracting *fundus uteri* with the hand, and maintain manual pressure until continuous tonic contraction is established; to be prudent and conservative in dealing with the third stage, whether delayed or not; to guard the patient against dangerous positions at the time of delivery, and against dangerous change of position or agitation afterward; in a word, and adopting the phrase of M. Hervieux, to "conform rigorously to all the precepts of the art."

I draw the following conclusions: The question being on the occurrence of fatal accidents from spontaneous entrance of air into the veins, those who take the affirmative can accept the burden of proof and urge the paramount importance, in every disputed case, of the *earliest possible* measurements and analysis of the gaseous contents of the veins and heart. This is what the opponents of the proposition have failed to do or even to suggest. Experimental work should also be directed to declare the mechanism of air access to the heart by the channels of the remoter vessels, whether they be the sinuses of the dura mater or the sinuses of the parturient uterus.

These are the problems which claim elucidation, to the end that the theory of accidental fatal air embolism be no longer dependent on testimony however strong, nor on analogy however convincing, but be established on an indisputable basis of demonstration.

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REVIEWS.

A System of Practical Surgery. By Prof. E. von Bergmann, M.D., of Berlin; Prof. P. von Bruns, M.D., of Tübingen, and Prof. J. von Mikulicz, M.D., of Breslau. Edited by William T. Bull, M.D., Professor of Surgery, College of Physicians and Surgeons, Columbia University, New York. Philadelphia and New York: Lea Brothers & Co.

The authors of this "System" have divided their subjects on a topographical basis, so that the surgery of the Head, the Neck, the Thorax and Spinal Column, the Extremities, the Alimentary Tract, and the Pelvis and Genito-urinary Organs is considered in five separate volumes under the above titles.

The book, in spite of the excellent work of Dr. W. T. Bull, the editor, and of the American translators, is in essence an exposition of Continental surgery, and would have no injustice done it if it were labelled "made in Germany."

Just such an exposition in English of German theory and practice is, however, at this time of distinct value to American practitioners and to teachers too busy to consult the original writings of the distinguished authors, although it should be regarded as supplementary to the standard English and American "systems" and text-books.

If any one body of surgical authors or practitioners were to be selected to-day as the chief authority, the court of last resort, in surgery it would not in the opinion of the writer be found anywhere east of Greenwich or west of San Francisco. It was, therefore, somewhat of a disappointment on reading this book to note the almost exclusive reference to German views, methods, statistics, and experiences. Of those views, methods, etc., it is, however, a most interesting, complete, and useful exponent, and, after all, there probably was never a time during the period covered by modern surgery—*i. e.*, the period since Lister introduced antisepsis—when there were so few radical differences as there are now among surgeons of differing nationalities.

In Volume I. the most important article is that on Injuries and Diseases of the Skull and its Contents, by von Bergmann and Krönlein. It is most instructive, and while it may now seem to err in the direction of conservatism, it is not improbable that its

somewhat dogmatic teachings may later become the accepted views of the profession.

The statement that in fractures of the base, the prognosis is more unfavorable than in fractures of the vault "because the accompanying brain lesion is deeper and more extensive," should be extended to include the greater risk of infection due to the impossibility of ensuring sterility of the nose, pharynx, and auditory canal.

The usual advice to trephine in cases of "funnel-shaped fractures," even although in so doing a simple fracture is converted into a compound one, is qualified by the statement that "whenever" such a fracture or a narrowly limited depressed fracture is combined with localized brain symptoms, the depressed, splintered, cone-shaped fragment of bone must be removed, whether in case of a simple or compound fracture; and further, by the remark that neither the authors nor other observers have ever seen a funnel-shaped depressed fracture occur without a wound of the soft parts.

The existence of localized brain symptoms, or of irritation in these cases, is emphasized because the authors believe that narrowly limited depressions never cause symptoms of compression of the brain, no matter how deep they may be, for "the diminution of intracranial space brought about by the projecting cone-shaped fragment of bone is never sufficient to disturb the circulation of the brain and its membranes." They add that "depressions limited to a small area have been held responsible for subsequent *epilepsy*, and to remove or elevate depressed areas of bone in every case has been considered positively indicated. As a matter of fact, epilepsy occurs as frequently in connection with head injuries in which no fracture exists. Depression is no more a factor in the etiology of this disease than any other scar of the brain or external soft parts that has become adherent to the skull."

Gunshot wounds of the skull are considered with sufficient thoroughness, but it is to be noted that the article dealing with them is based upon the work of Kocher and von Bruns, and upon that of von Coler and of Schjerning in the "Imperial Royal Prussian War Department." No reference is made to the British or American experiences in the South African and Spanish wars.

"Undubitable," page 87, and "acustic," page 92, may be intentional, but are probably typographic slips.

In Volume II. one of the most interesting of the articles is that upon Goitre, by von Eiselsberg. The author believes that medicinal treatment in goitrous cases should at present consist exclusively in the use of iodine, and adds: "The action of this drug is certain. According to Kocher, 90 per cent. of all goitres may be improved by the use of iodine to such an extent that operation will be unnecessary. The efforts of pharmacology are not directed to finding a substitute for iodine, but to the use of the best preparation." He

includes the treatment of goitre with thyroid substance, or with preparations of it, as a modification of the iodine treatment, because Baumann's experiments have shown that iodine is contained in the active principle of the thyroid gland. He cites the experience of von Bruns, who administered 5 to 15 grains of the gland daily in the form of tablets to 350 patients, and reported 8 per cent. of complete cures, 36 per cent. of good effects, 30 per cent. of moderate success, and no success in the remaining cases. When he adds, however, that a number of authors report cases in which this treatment was productive of no result whatever, he might safely have included the large majority of American surgeons in whose hands the thyroid treatment and the iodine treatment have been exceedingly unsatisfactory.

A no less interesting article on the Thorax and its contents, by von Riedinger and Kümmell, deserves mention for its thoroughness and clearness.

In the section on Carcinoma of the Mamma, the noticeable increase of cancer in all countries is commented on; and in Prussia the death rate from this disease is said to have increased 153 per cent. since 1877. The authors do not believe that local recurrences after operation are due to infection with cancer cells at the time of operation, although they do not deny that this may be possible. In considering the improved results of recent years—unquestionably due to earlier as well as to more extensive operation—not enough emphasis is placed on the former factor, which is the more important of the two.

It is interesting to note the statistics of so-called permanent cures —*i. e.*, cures in which three years have elapsed without recurrence. These vary from 19.4 per cent. (Horner) to 42 per cent. (Rotter, Heidenhain). In the writer's opinion the three-year period, while convenient as an arbitrary basis for description of results, is illusory if considered as justifying the literal use of the term "permanent cure." The teaching that cancer of the breast in its early stages is a local disease, that a permanent cure is possible by radical removal of the tumor, and by that method only, that the condition never gets well of itself, and that the value of other methods of treatment is extremely doubtful, would be subscribed to by the vast majority of surgeons everywhere. Thorough and radical operation is recommended, but the methods in which all the infra- and supraclavicular glands are removed, suggest to the authors the remark that it is doubtful whether the success in operations of this sort is proportionate to the severity of the interference, because in these cases the intrathoracic lymphatics are almost certain to be diseased. Heidenhain's and Stiles' investigations have shown how very much more extensive lymphatic infection may be than the evident metastases would seem to indicate. "In carcinoma with involvement of the clavicular gland therefore the operation in

all probability will be done too late, and a permanent cure extremely doubtful."

On page 301, parenchymatous "stroma" should be "struma."

In Volume III., devoted to the surgery of the extremities, occurs perhaps the most conspicuous example of the inconvenience of the topographical division of the subjects. Fractures, for example, are not found as a separate section, but must be hunted up under the surgery of the various regions. Such inconvenience is necessary in works on applied anatomy, but is objectionable in surgical text-books.

As a rule, surprisingly little difference is to be observed in the apparatus recommended in the treatment of these injuries. It has not been long since a German text-book in treating of fractures of the thigh would have devoted pages to cumbrous and antiquated splints. Here, Hoffa, while mentioning the exceptional cases in which special apparatus is required, says "the various immobilization splints and apparatus formerly used often did more harm than good, and are replaced at the present time by position-, strip-, and plaster-splints and continuous weight extension." If, however, any mention is made in the consideration of ununited fracture of this and other bones of the most valuable mechanical addition to our methods of treatment—Parkhill's splints—it has escaped the notice of the writer. For example, in discussing pseudarthrosis after fracture of the shaft of the ulna, Wilms says that of the various methods proposed to stimulate ossification, he "will mention only the insertion of ivory pegs and suture of the bone," both of which have been found by surgeons in this country to be relatively very unsatisfactory except in cases of extremely oblique fracture where the two fragments may readily be transfixed. Even then a simple screw or gimlet is superior to either of them.

In the article on Amputations no description is given of what many surgeons regard as the best method of performing what is probably the most frequent of all amputations—the amputation of the leg—the method which secures a periosteal flap without disturbing the relations of the deeper structures. The methods of Bier and Storp involve the retention of a "bone hinge plate," which is an unnecessary and clumsy complication of the operation. Unqualified approval may be given to the statement that if the surgeon is uncertain as to whether the tissues are infected or not, or if speed is required in severe accident cases, "it is urgently recommended not to close the wound, but to pack with sterile or iodoform gauze after checking the bleeding, to apply a dressing with slight pressure, and to close the wound secondarily."

In discussing the prognosis of amputation of the arm, the same writer says that the mortality in uncomplicated cases is "practically *nil*." This favorite but slovenly expression is objectionable. In this, as in other surgical procedures, either there *is* a mortality, in

which case it is not "*nil*" whether viewed from a "practical" or from a theoretical standpoint; or there is *no* mortality, which fact is susceptible of clear statement in English.

On page 163, the legend under Figure 103 describes it as an "*X*-ray of Figure 103." It should probably be of "Figure 102." On page 541, "counterindicated" should, we think, be "contra-indicated," although the former word is used throughout by the majority of the translators.

Volume IV. contains an exhaustive series of articles on the Surgery of the Alimentary Tract. In the article on the treatment of hernia (Graser), Czerny's operation is first mentioned and is said to be the simplest. Bassini's operation is imperfectly described, and is illustrated by somewhat clumsy wood-cuts, and rendered more confusing by the description which in Figure 229 begins "Operation for the cure of oblique inguinal hernia," and in Figure 230, shows the "strangulated contents" of the sac.

Halsted is not mentioned. Various operations now regarded by most surgeons as obsolete, such as Macewen's, are described with no expression of opinion.

The statistics of operations for radical cure would seem to show only about 10 per cent. of recurrences. It is very doubtful whether this is correct, the recorded results varying greatly with the age of the patients, their general health, their subsequent occupation, and the time at which the report is made.

Schlange's operation on Appendicitis is of special interest. Two of a series of rules which he gives may here be quoted: "There is a relatively large group of cases of appendicitis in which the attack is from the start of slight character. Since the general condition of the patient is little affected, there is not much infiltration of the tissues and the fever is slight or becomes so by the third day. In such a case an operation is counterindicated, although the patient or his nurse should be advised that it may become necessary at any time. Furthermore, one should point out the probability of recurrence so that operation in the interval may be performed if desired."

"Whatever the skill of the operator, he should not insist upon removal of the appendix if it is so situated that its removal will greatly complicate the operation."

These are singled out because of their relation to questions still under dispute. Unequivocal disapproval should be given to the teaching to give the patient in the early stages, "before the diagnosis is certain," "a moderate amount of opium, and if necessary, morphine to control the pain." He adds: "Cathartics and large rectal injections are inadvisable since they stimulate peristaltic action and may drive fluid fecal matter through the perforated appendix." This has been abundantly shown to be a purely imaginary danger. The reviewer has never seen a case of death from appendicitis

which could be remotely attributed to the administration of laxatives in the early stages. He has seen many such cases in which the fatal result was due to the use of opium.

Volume V. deals with the Genito-urinary Tract and the Pelvic Organs. As a rule, the teaching is sound, but in some places seems to be antiquated. The statement that "suprapubic cystotomy should be chosen for the removal of vesical calculus in a boy under fourteen years of age" cannot be allowed to pass without question. It certainly does not represent the views of English or American surgeons at this time.

Freyer's operation for the total removal of the prostate is not mentioned, and the author says: "A study of the anatomy of prostatic hypertrophy makes it somewhat doubtful whether one ought ever to attempt to remove an enlarged middle lobe of the prostate through the bladder!"

As to the "sexual operations" for enlargement of the prostate, he says truly that "it is fair to conclude that sexual operations," meaning thereby both castration and substitutes for it, "have not fulfilled the hopes of their originators;" and adds that "in view of the surrounding circumstances, and of the imperfect results thus far obtained, it may be stated that sexual operations yield no better results than other operative measures, and that, therefore, they should only be performed under special conditions."

Why he should have classed ligation of the internal iliac arteries with the sexual group of operations it is difficult to say.

The article by Schede on the examination of the kidney and ureter is a very practical and instructive one, and has been brought well up to date.

Steinthal's article on the Surgery of the Rectum is another one well deserving of favorable mention. In carcinoma, he regards early operation as determining the degree of success which may be obtained. In prolapsus, he recommends cauterization of the superfluous membrane. In the treatment of hemorrhoids he compares excision and cauterization, and describes the use of the ligature in only a few lines, adding that "it is claimed that suppurating thrombophlebitis and pyæmia may follow this operation." This is a very unfair presentation of the case. His own figures show that while Allingham has treated 1600 patients by this method without a fatality, Mikulicz has lost after excision two cases out of one hundred and thirty.

The general appearance of the book is most pleasing; the illustrations are numerous and usually adequate to the elucidation of the text; the rendering into English has been admirably done and reflects credit on the scholarship of the translators; Dr. Bull's name is sufficient evidence of the excellence of the editing.

If the publishers instead of saying "the special features wherein American surgery differs from that practised in Europe are fully

reflected," had said that the points wherein German surgery differs from that practised in America were fully set forth; and if instead of saying that "these five volumes constitute a complete working library on Surgery," they had said that no working library on Surgery could be considered as complete without them, the reviewer would cheerfully have subscribed to their not otherwise extravagant commendation of this most excellent "System." J. W. W.

EPILEPSY AND ITS TREATMENT. By WILLIAM P. SPRATLING, M.D., Medical Superintendent of the Craig Colony for Epileptics; Secretary of the National Association for the Study of Epilepsy and the Care and Treatment of Epileptics; Member of American Medico-Psychological Association, New York Academy of Medicine, Buffalo Academy of Medicine, Rochester Pathological Society, American Medical Association, etc. Fully illustrated. Philadelphia, New York, and London: W. B. Saunders & Co., 1904.

THIS volume of 500 pages treats the subject of epilepsy thoroughly from every point of view. Coming from the pen of one with Dr. Spratling's experience with this disease it is sure to fill an important place in the literature upon epilepsy. The method employed by the author is to give a review of the literature followed by an account of his own experience in each topic discussed, showing where his own researches substantiate or disagree with the ideas generally held. Many cases are used for means of illustration.

The subject is treated under the usual heads of Definition, Classification, Etiology, Diagnosis, Prognosis, Pathology, Treatment and Psychological and Medicolegal Aspects, and the arrangement of the book is excellent. It should prove very satisfactory as a book of reference, tables and classifications being numerous.

The chapter on Status Epilepticus has been written by L. Pierce Clark, M.D., who has, together with Thomas P. Prout, M.D., written the chapter on the pathology of epilepsy. Both of these chapters have separate bibliographies. In the latter the authors largely base their opinions on investigations carried on at the Craig Colony for Epileptics, and consider that the disease may be regarded as a highly organized sensory motor reflex of the cerebral cortex. The chapter on prognosis is of interest in that it takes up the importance of various conditions which mark cases as curable or incurable. Spratling's statistics show that at the Craig Colony 5 per cent. of the 643 possible curable cases have recovered.

The author divides treatment into general, medical, and surgical, and under the first division lays special emphasis on the Colony

method of treatment, where not only the disease but the individual is recognized and treated, a factor which he considers very important.

The book is if anything too comprehensive, and includes some matter that could be omitted. Such is especially noted in the chapter on Surgical Treatment, where detailed descriptions of the technique of trephining and abdominal section are gone into. The literary style is straightforward and clear. As it is the first American book to appear upon the subject of epilepsy for thirty-three years its appearance should be welcomed. Treating as it does such an important disease in a very lucid and thorough manner it is sure to be of much value. C. C. R.

A Treatise on Materia Medica and Therapeutics, including Pharmacy, Dispensing, Pharmacology, and Administration of Drugs. By Dr. R. Ghosh. Edited by Lt.-Col. C. P. Lukis, M.B. Calcutta: Hilton & Co.

Although this work is primarily intended for East Indian students and physicians, it is nevertheless one which possesses its value for readers in other countries. The material contained in it is so arranged as to make it of the greatest practical value as a ready reference handbook. There are several novel features which appeal strongly to the practising physician, among which we would particularly mention what the author terms "prescribing hints," which tell us in what manner the various remedies may be most elegantly prescribed. These hints are appended to the description of the more important drugs. The work follows the British Pharmacopœia, but has in addition a very complete account of the medicinal preparations which are contained in the Indian edition to that work. Many American authorities would do well to adopt the excellent classification and practical arrangement which is found throughout this book. J. N. H.

Normal Histology. By Edward K. Dunham, Ph.B., M.D., Professor of General Pathology, Bacteriology, and Hygiene in the University and Bellevue Hospital Medical College, New York. Third edition, revised and enlarged. Illustrated with 260 engravings. New York and Philadelphia: Lea Brothers & Co., 1904.

The present edition of this book is to be highly recommended. Short, concise and clearly written it forms an excellent supplement to the student's practical work in this branch of his medical educa-

tion. Since the volume is not intended as a book of reference the reader is referred to larger treatises, and anatomical journals for a more extensive survey of certain subjects which cannot here be treated in a broad manner. The main facts and important principles concerning the cells, the tissues, and the various organs are very clearly stated. The ductless glands, so grossly ignored in the older text-books, receive appropriate space. The histology of the nervous system is dealt with in an elementary manner but this cannot be considered as a fault, since the subject is too large to admit of extended treatment except in a volume devoted especially to this phase of anatomy. The organs of special sense, too, are described in 12 pages. The final chapter on Histological Technique scarcely seems necessary for books devoted exclusively to this subject are readily obtainable for class-room work and give a much more extensive view of technical methods than can be put into the short space used by the author. Most of the illustrations are good, and the cuts adapted from other text-books have been, as a rule, well chosen. W. T. L.

NERVOUS AND MENTAL DISEASES; A MANUAL FOR STUDENTS AND PRACTITIONERS, WITH AN APPENDIX ON INSOMNIA. By JOSEPH DARWIN NAGEL, M.D. Philadelphia and New York: Lea Brothers & Co., 1904.

THIS is a good little book for a student to use to prepare for a quiz or to work up for a theoretical examination. It is of the class that a conscientious teacher regards with more or less justifiable suspicion and that the student utilizes as a very present help in trouble. It is much better than the quiz compends, because it gives coherent and, so far as is possible in such limited space, accurate descriptions of diseases. There is, unfortunately, owing to our methods of teaching, a period in the life of every medical student when he must learn certain things by mere force of memory and without the use of reason. For that period this book is a very good one. For the practitioner it will be found a convenient compendium for ready reference. C. W. B.

CLINICAL LECTURES ON MENTAL DISEASES. By T. S. CLOUSTON, M.D. Edin., F.R.C.P.E. Sixth edition. Philadelphia and New York: Lea Brothers & Co., 1904.

A BOOK that has passed through six editions, and has been before the public as long as this, little needs reviewing. It has proved its worth. We must say, however, that it is a great pleasure in these

days, when so many books are merely manufactured like shoes or sausages, to read one that has really been written, and that by a man who speaks from his own knowledge and pays attention to the rules of grammar. Clouston is somewhat old-fashioned. For example, the word paresis does not occur in the index, the term general paralysis alone being used. Paranoia, which in most books of to-day occupies many pages, here has little more than two, but much concerning it is discussed under monomania. Maniac depressive insanity is not mentioned in the index, and nothing is said about the association of insanity with certain types of neuritis. We mention these omissions not in disparagement of the book, but to show its scope and the point of view of the author. The book is most interesting and valuable and gives excellent pictures of insanity. This edition differs from the earlier ones very markedly in the sections on pathology. C. W. B.

THE DOCTOR'S LEISURE HOUR. FACTS AND FANCIES OF INTEREST TO THE DOCTOR AND HIS PATIENT. By CHARLES WELLS MOULTON, General Editor. Arranged by PORTER DAVIES, M.D. Chicago, Ohio, and New York: The Saalfield Publishing Co., 1904.

THIS book, compiled by a doctor in his leisure hours for the amusement of other physicians in theirs, contains a curious hodge-podge of jokes, stories, and poems gathered from newspapers and other periodical literature, intermingled with a few borrowed from the literature of a higher class; thus we find extracts from the works of Molière and Oliver Wendell Holmes mingled with jokes and poems such as pass current for wit in the pages of our daily newspapers. Many of the anecdotes have figured in other collections of professional ana such as "The Gold-Headed Cane," and Jeffries' famous "Book about Doctors." Although there are many extracts in the collection which reflect credit upon the profession, nevertheless we doubt if it is wise from a physician's point of view that such a collection should ever fall into the hands of the laity. It is bad enough to be compelled to read the cheap witticisms on appendicitis, faith cure, and Christian Science which are now so current in the daily press, without having them gathered in a quasi-respectable form. It is good that such collections of medical ana should be published occasionally, as there is much gold among the baser metal which they contain, and we think this book will probably achieve, as it deserves, a very considerable popularity in the ranks of the profession. F. R. P.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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Gonorrhœal Phlebitis.—JULIUS HELLER (*Berliner klin. Wochenschrift*, 1904, xli., 609) reports a case of gonorrhœal phlebitis of the posterior saphenous vein and of the pampiniform plexus of veins on the left side coming on four weeks after all urethral symptoms of a rather severe attack of gonorrhœa had subsided. He further reviews 25 cases he has been able to collect from the literature; 20 of the cases were in men, 6 in women, and all but 2 occurred in the decade from twenty to thirty years of age. In all but 1 case the complication accompanied the first attack of gonorrhœa. The phlebitis usually occurs in the subacute stage of the urethritis, the average period of incidence in fifteen observations being four and one-half weeks after onset. In 1 case it came on fourteen days after onset, and in 3 others three, sixteen, and eighteen months after, respectively. The original attack of gonorrhœa may run an uncomplicated course, but it is more often severe and associated with complications—prostatitis, urethrocystitis, epididymitis, pleuritis, pyelitis, and erythema nodosum being reported; 15 of the cases were accompanied with arthritis.

The veins affected were as follows: the common iliac (side not mentioned), 1; the internal iliac (side not mentioned), 1; the femoral: right, 3; left, 3; the popliteal (side not mentioned), 1; the deep femoral: left, 2; side not mentioned, 1; the internal saphenous: right, 9; left, 6; side not mentioned, 1; superficial abdominal veins (side not mentioned), 3; vaginal veins: left, 1; dorsal vein of penis, 1; vein to the corpus cavernosum, 3; prostate and vesical veins, 2; pampiniform plexus: right, 1; humeral vein: right, 3; left, 1; radial and cubital veins: left, 1. In most cases a single venous system is affected; in only 6 cases separate systems. Fever was present in 10 cases, in 1 continuous and resembling somewhat a typhoid curve. In 2 cases chills were noted. The outcome of the phlebitis is usually a complete *restitutio ad integrum;* this was the case in 16 cases—62 per cent. The duration in these cases

varied from a few days to ten weeks, with an average of six weeks in ten observations. Occasionally, however, the cure is not so complete. In Empe's case the vein was still obliterated and œdema present after three months, and in the cases of Monteux and Lops there was persistent induration of the affected vein. In Hasemud's case amputation of the thigh was necessary. Pollard's patient died of pulmonary embolism.

Heller ascribes phlebitis in these cases to the gonococcus, although the fact has not been established anatomically. He suggests that in the cases with high fever and marked constitutional symptoms there may be a mixed infection.

Dysbasia Angiosclerotica (Intermittent Claudication).—W. ERB (*Münchener med. Woch.*, 1904, li., 905) reports 45 cases of this interesting condition as a supplement to his original publication in the *Deut. Zeitschr. f. Nervenheilk.*, 1898, xiii., 1–76.

In 38 cases the condition was typical—intermittent exhaustion on walking, with rapid resuscitation after rest; in 7 cases atypical, the intermittent claudication being replaced by symptoms only suggestive, as weakness and rapidly manifested fatigue, pain in the legs on exercise, with paræsthesias and sensations of cold. In all of these cases, however, there was the characteristic complete or incomplete obliteration of the pulse in the arteries of the feet and well-marked general arteriosclerosis. In 30 cases the condition was bilateral; in 15 unilateral, with marked selection of the left side—11 to 4. Of the 30 bilateral cases all four of the pulses were absent in 16 cases; three in 2 cases; two in 7 cases; one in 1 case. In the remaining 4 cases the pulse was palpable, but very weak. Of the 7 unilateral cases both pulses were absent on the affected side in 5 cases; one absent and the other weak in 1 case, and both palpable in 1 case. In 2 cases both of the pulses were absent even on the apparently healthy side, and in 4 more one pulse was absent on the symptom-free side. In some instances the popliteal pulse was obliterated, and in a few even the femoral.

The associated general arteriosclerosis was variable. In 8 cases it was only slightly marked or even absent, while in the remaining 37 it was very evident in the peripheral arteries, varying from moderate to most extreme grades. None of the cases were associated with granular kidneys—at least in only one was this a possibility—and only 2 showed albumin in the urine. Cardiac changes could be demonstrated in but 15 cases, the changes consisting in an accentuated second aortic, a systolic murmur, slight left ventricle hypertrophy, an irregular or intermittent pulse, and in 1 case attacks of angina pectoris. Vasomotor disturbances—cold feet, pallor, flushing, cyanosis, etc.—were observed in 32 cases, absent in 11. Two cases had gangrene. Neurasthenia was a prominent symptom in 5 cases, but the examination of the nervous system was normal in all 45, excepting 1, in which the Achilles tendon reflex was absent, and a concurrent spinal affection could not be definitely excluded. Erb has met with cases in which, with typical symptoms of intermittent claudication, the arteries were apparently normal and the pulses well felt. These he refers to as purely neurotic (vasomotor?) forms of the affection. On the other hand, patients are encountered with entirely or almost absent pulse in the tibial arteries who are perfectly free from symptoms.

All of Erb's 45 cases were males, and of 81 cases collected from the literature only 6 were females. The age of the patients at the onset of symptoms was: under thirty years, 1 case; from thirty-one to forty years, 7 cases; from forty-one to fifty years, 16 cases; from fifty-one to sixty years, 12 cases; from sixty-one to seventy years, 9 cases. Previous syphilitic infection was present in 10 cases—22.77 per cent. This cannot be considered of etiological moment, as in the statistics of 10,000 men over twenty-five years of age Erb found a history of syphilis in 21.5 per cent. 32 of the cases used alcohol moderately or not at all; 7 cases admitted excesses, and in 6 there is no note of the habit. 15 cases gave a history of excessive smoking, and 10 of heavy smoking; 13 smoked moderately or not at all, and in 7 cases the relation is not noted. Erb is inclined to lay much emphasis on the abuse of tobacco as an etiological factor. 12 cases gave positive history of exposure to cold, 6 of these being inhabitants of Russia; 2 cases had gout, 2 diabetes, and 1 lead-poisoning. As possible influencing factors are further mentioned the abuse of coffee and continuous pressure on the femoral artery by a truss for the control of rupture.

[The reviewer has now under observation a most typical example of intermittent claudication of the left leg following an acute arteritis with probable parietal thrombosis of the femoral vein in typhoid fever. Reference may be found to the case in an article on the "Cardiac and Vascular Complications and Sequels of Typhoid Fever," in *Johns Hopkins Bulletin* for October, 1904.—W. S. T.]

The Occurrence of Herpes Zoster in Croupous Pneumonia.—Dr. Riehl (*Münch. med. Woch.*, 1904, li., 1105) states that in 481 cases of croupous pneumonia herpes was noted in 129 cases, or 26.82 per cent. This is a little below the figures usually quoted, Drasche giving 40 per cent.; Metzger, 43.2 per cent.; Smaler, 32 per cent. Of the 129 cases 99 were males and 30 females. The occurrence of herpes is most common in robust patients in the second and third decades, and notably infrequent in the very young and very old. Following the usual rule the eruption occurred most often on the third, fourth, and fifth day of the disease, followed soon after (two to four days) by the crisis; not infrequently, however, it appeared on the second or was delayed until the ninth or tenth day, and in one instance it appeared on the twenty-sixth. Recurrence of the eruption during convalescence, as was reported by Bleules in 9 cases, was not noted by Riehl. The pneumonia proved fatal in 70 out of 481 patients, and of these 70 only 6 had an eruption of herpes. This distinctly emphasizes the favorable prognostic significance of the eruption, a significance previously insisted upon by many observers. Herpes is usually most abundantly developed in the mildest cases of pneumonia. The figures in reference to the distribution of the herpes are interesting enough to quote in full. In 88.36 per cent. of the cases the eruption occurred in the region of distribution of the second or third branches of the trigeminus. The upper lip was involved in 25 cases; upper and lower lip, 24; upper lip and angle of mouth, 1; upper lip and nose, 7; upper lip, under lip, and nose, 3; upper lip, angle of mouth and nose, 2; upper lip and chin, 1; upper lip, under lip, and chin, 1; lower lip, 16; lower lip and angle of mouth, 4; lower lip and nose, 2; nose and nasal septum, 15; angle of mouth, 8; angle of mouth and chin, 1. The eruption occurred on the same side as the

pneumonia twelve times; on the opposite side twelve times. In 46 cases of right-sided and 39 cases of left-sided pneumonia it was localized on both sides; in 17 cases of double pneumonia it occurred three times on the right side; three times on the left side, and eleven times on both sides. As unusual localizations, herpes may be mentioned on the tip of the tongue, once; on the ear, three times; on the forehead, twice; and on the upper eyelids, twice.

SURGERY.

UNDER THE CHARGE OF

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AND

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The Technique of the Use of Venous Hyperæmia.—HENLE (*Centralblatt für Chir.*, 1904, No. 13) states that in the use of venous hyperæmia one has a valuable antidote against tuberculosis of the extremities, as has been clearly shown by the experiments that have been conducted in the Breslau Surgical Clinic. That it also is effective in either chronic or acute affections of the joints has been proven by many experiments. Its value in erysipelas has been well shown in many cases. Bier found as the result of his experience that the best results are to be obtained by only producing a hyperæmia for the space of one hour daily. Care should be taken that the arterial circulation is not interfered with, and the best results have been obtained not by using the Esmarch bandage, but by the use of a band similar in its construction and application to that which is found as a part of the Riva-Rocci blood-pressure apparatus. By the use of this special apparatus, which the author has designed, the pressure can be most accurately regulated, and the degree of venous hyperæmia easily controlled.

The Diagnosis and Treatment of Hemorrhoids.—BRICK (*American Medicine*, July 23, 1904) after an admirable and most complete review of the surgical treatment of this most common condition states in conclusion that the method selected should be the one best suited to the individual case, although no case of internal piles that has come under the author's observation has been too severe for the clamp and cautery method. There is a double protection against hemorrhage both by the crushing action of the clamp on the tunica intima and by the cauterization, the thermic action of the cautery destroying all septic germs which may be present on the operative field; all capillaries and lymphatics are sealed, which prevents absorption of septic agents; there are no ligatures to cut through; the operation is finished at one time; no

protracted inflammation from injured nerves; no sutures to be removed, and there is a gain of about one week in the time of healing.

The Treatment of Chronic Bright's Disease.—ROVSING (*Centralblatt für Chir.*, 1904, No. 17) states that in a previous publication Edebohls has misquoted some statements and attributed them to him, and with a view to clearing up the facts it becomes necessary to again go into the question in detail.

Edebohls has stated that in his opinion there should be total extirpation of the membrana propria if the kidney and its capsules are either partially or totally diseased, so that there may be union between the surface of the kidney and its surrounding fat capsules.

Rovsing entirely disagrees with this view, for the reason that when this is done the kidney becomes embedded in a mass of newly formed connective tissue which, when new, guarantees an ample supply of blood, but later these adhesions of necessity retract and atrophy, and, as a result, the blood supply to the kidney is distinctly impaired and the kidney consequently undergoes atrophy. In Rovsing's opinion capsulectomy is only indicated in those cases where the kidney is either partially or totally bound down by adhesions, or if the kidney be tense and markedly swollen, or the membrana propria is distended by cysts. Such a condition is more often found in those cases where there is either a nephritis complicated by infection, or in nephrolithiasis, or in those cases which may be described under the name of a curative aseptic interstitial nephritis and perinephritis, and which may be recognized by pain in the region of the kidney, and in which pain rarely may be accompanied by hæmaturia. The pain in these cases is due to an irritation of the membrana propria, which is supplied by large numbers of sensitive nerve filaments. In Rovsing's judgment operation is only indicated in cases of nephritis dolorosa. In those cases where marked toxic conditions exist operation is not only not indicated, but in the majority of instances is extremely dangerous. In opposition to these views, Edebohls has taken the stand that in every case of chronic Bright's disease, no matter in what condition it may be, both kidneys should be subjected to decapsulation. Rovsing insists that in his judgment this is so grave an error as to require emphatic protest from the surgical as well as the medical point of view. It may be laid down as a broad general rule that each case must be considered upon its merits, and no case should be subjected to operative interference until it has been subjected to the most careful examination. Every case is not a suitable case for operation. It is to be regretted that a search through the literature shows that in the reports on the surgical treatment of Bright's disease no mention is made in many cases that there has been the careful and thorough examination, which should be the invariable rule before subjecting a patient to all the risks of operation. No one has been more to blame for this than Edebohls, as may be clearly seen by a reference to his article in the *Medical Record*, December 21, 1901. And a further examination of this article will show that out of eighteen cases not one of them has been subjected to a cystoscopic examination of the bladder with catheterization of the ureters and a subsequent bacteriological examination of the fluid obtained. The history of these cases is also lacking in many things, such as the exact amount of albumin the urine contained, and also the results of a careful micro-

scopic examination of the urine after sedimentation. The diagnosis in many of these cases would seem to have been obscure, and it appears to have been based upon two points: First, albuminous urine; and, second, inspection of the kidney through the small lumbar incision of Simon; but when one considers how often the floating kidney is complicated by albuminous urine from purely mechanical or infectious conditions, besides in many cases being found to be either atrophied or swollen, or the membrana propria adherent by reason of traumatism, it makes one extremely doubtful in the view of these facts as to whether or not a correct diagnosis had been arrived at in all of the cases which Edebohls subjected to operation; and this skepticism becomes of necessity more marked when one becomes aware of the fact that Edebohls reports that in nine out of these sixteen cases the other kidney was absolutely healthy, the Bright's disease being merely confined to the one side; and the subsequent note that immediately the kidney was sutured the albumin disappeared. Of the eight patients whom Edebohls reports as cured, in seven of the cases only one kidney was affected.

There is only one conclusion that can be reached in view of these facts, and that is the albuminuria has been due simply to the undue mobility of the kidney, and this has been relieved by suturing the kidney in its proper position in the loin. In only two of the cases was decapsulation performed. Neither of these patients had Bright's disease, and the result was that one of them died and the other is reported as not cured. It is of interest to see Edebohls' further reports upon this subject. In the *Medical Record* for March 28, 1903, he notes that at the end of the year 1902 he had operated upon not less than fifty-two cases of chronic Bright's disease, and that of these cases eleven died and not a single one was cured. It would, therefore, seem perfectly proper after due consideration to intimate that Edebohls has not given to the profession a single shadow of proof that chronic Bright's disease can be cured by operative interference.

Out of ten cases of chronic Bright's disease which Rovsing treated by decapsulation, there is not a single case in which a cure has been the result, and common-sense would seem to show that when one considers the pathology of the disease operative interference will be of no avail. Too much stress cannot be laid upon the fact that in Rovsing's judgment operative interference is absolutely contraindicated, except in those rare cases of chronic Bright's disease which are accompanied by severe kidney pain; and also in those cases of nephritis which are either traumatic, infectious, or dependent upon the irritation of uric acid for their origin. Therefore, it is absolutely essential that every case should be subjected to a most prolonged examination in order that the etiology of the nephritis may be clearly determined before any operation is decided upon. Too much stress cannot be laid upon the great importance of this careful early examination, which should include microscopic and bacteriological examination of the urine obtained by catheterization of the ureters.

The Treatment of Fractures of the Lower End of the Femur by the Bardenheur Method.—GRAESSNER (*Centralblatt für Chir.*, 1904, No. 11) states that the splendid results of this method in the treatment of supracondyloid fractures of the humerus has induced him to try this method in those fractures of the femur in which there is marked

deformity. Experience, which has been confirmed by *x*-ray pictures, shows that the deformity can be completely overcome by a weight of 25 kilograms in men and 20 kilograms in women. The subsequent examination of the patients who have been subjected to this method of treatment has shown that good union is secured without the shortening which seems inevitable under other methods of treatment.

THERAPEUTICS.

UNDER THE CHARGE OF

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Hyperchlorhydria and its Treatment.—DR. B. H. TRAGO believes that hyperchlorhydria is a disease, not a symptom. In treatment we should first prescribe a milk diet. Antiseptic medication causes a cure in certain cases. The physician should remember how the disease develops and the therapeutic indications which may arise. Gastro-intestinal anastomosis is a last resort. This operation is not applicable in the treatment of all cases of hyperchlorhydria with pyloric spasm. Lastly, the mixed medical treatment is the one which will result in the largest number of cures.—*Revue de thérapeutique*, 1904, No. 15, p. 525.

Dechloration in Hysteria.—M. VINCENT is of the opinion that sodium chloride in excess not only affects the stomach and kidneys, but aggravates the tendency to hysteria in patients who tend to high nervous excitability. If hysterical persons are given three to four drachms of salt per day, in addition to their regular allowance, the nervousness is increased and the number of hysterical seizures augmented. Likewise, salt appears to excite persons affected with latent hysteria. At the same time gastric disturbances, insomnia, and other signs of hysteria become more apparent. On the other hand, upon a diet lacking in salt these symptoms tend to disappear. Thus, salt in excess plays the same part in hysteria as alcohol, lead, mercury, etc., and exaggerates any hysterical tendency. In patients who are affected with hyperchlorhydria as well, diminishing the chlorides will act favorably upon the stomach as well as upon the system in general.—*La semaine médicale*, 1904, No. 28, p. 221.

The Choice of an Acid for Gastric Medication.—DR. ALFRED MARTINET, after a discussion of the various acids that may be employed, concludes that hydrochloric and phosphoric are the only ones admissible; he is strongly in favor of the latter, since it possesses the following advantages: It does not lessen the secretion of hydrochloric acid in the stomach. It is quickly neutralized in the intestine and changed into sodium phosphate, a normal constituent of the blood. It is elim-

inated, at least in part, by the kidneys as acid sodium phosphate, a normal constituent of the urine. It lessens the acidity of the urine and contributes toward rendering the urinary tract aseptic. It acts on the nervous system as a tonic, especially in dyspeptics, and the results achieved by its use justify its employment. The author gives the following formula: official phosphoric acid, 10 parts; acid sodium phosphate, 20 parts; water, 200 parts. Dose, one-half to two teaspoonsful in a glass of water, wine, or beer, to be drunk during the mid-day and evening meals. Its administration may be continued for long periods without the least bad effect.—*La presse médicale*, 1904, No. 59, p. 469.

Gastric Lavage in Infants.—Dr. J. Cassel states that gastric lavage has limited indications in pediatric practice. It is, however, especially useful in gastric atony caused by inappropriate feeding, particularly when the food is too watery or too abundant. The child should be fed at intervals of about four hours, and three hours after a feeding, the quality and quantity of which have been carefully noted, what remains in the stomach is drawn off. The procedure is kept up until the food has been so altered that none is left three hours after a feeding. Another indication is the diarrhœa and vomiting which affects infants recently put upon artificial feeding. Contraindications to lavage are collapse, inflammations of the respiratory passages, laryngitis stridulus, eclampsia, and tetany.—*Fortschritt der Medicin*, 1904, No. 18, p. 767.

An Antitoxic Treatment of Tuberculosis.—Dr. E. Wahlen concludes a paper upon this subject thus: The treatment of tuberculosis by tuberculous iodonuclein gives brilliant results when administered at the beginning of the formation of anatomical lesions. If the invasion of the tissues has not been present more than a few months an *apparent* cure may be achieved. From six to twelve injections will accomplish this result. In advanced tuberculosis, where surgical ablation of the diseased tissues is impossible, the recent lesions are unaffected; however, in such cases the resisting powers of the organism are not sufficient to withstand the effects of so active a treatment. The author injects a solution which contains to each fifteen minims: nuclein, $\frac{5}{100}$ of a milligram; iodine, $\frac{1}{1000}$ of a milligram; potassium iodide and sodium borate, of each, $\frac{2}{1000}$ of a milligram. The dose for an adult is 5 minims at first, gradually increased to 20; for an infant 1, gradually increased to 10 minims. The injections are painful, but produce no inflammatory reaction.—*Gazette médicale de Paris*, 1904, No. 29, p. 338.

Intrapulmonary Infusions.—Dr. Rosenheim advises the following method of procedure: The pharynx and larynx are cocainized, and a tube, through which are injected several cubic centimetres of 2 per cent. eucaine solution mixed with a few drops of 1:1000 adrenalin chloride, is introduced into the trachea. Thus a tube may be passed into a bronchus without provoking a reflex act. Cough appears at the moment that the eucaine is injected, but lasts but a moment. If there is tendency to hæmoptysis the procedure is contraindicated. Through the tube introduced into the bronchus five to seven drachms of 0.01 per cent. tuberculin solution are injected. The injection is repeated in three or four days, and if no reaction is observed the strength of the solution is

increased gradually to 0.1 per cent. The tube is of small calibre, and about sixteen inches long. The patient should breathe deeply during the operation and for some time afterward if the solution is to reach the apex of the lung. DR. JACOB reports that five patients at the beginning of the second stage of the disease, who had been ill for about a year and were much emaciated, to whom from fifteen to forty treatments had been given during from three to six months, were much improved. From four to eight weeks after the beginning of the treatment the bacilli disappeared, the body weight was increased, and a considerable diminution in the extent of the physical signs was noted. —*Revue de thérapeutique*, 1904, No. 15, p. 526.

Adrenalin in Cancer of the Upper Respiratory Passages.—M. ICHARD observes that topical applications of adrenalin, while they may not effect a cure, sensibly diminish the pain and tendency to hemorrhage of epitheliomata. He has obtained especially good results in cancers of the pharynx and larynx. In certain cases an almost complete cicatrization has been brought about, which in one patient lasted ten months. The 1:1000 solution was first employed, but on account of its deterioration upon standing the following formula was advised: Adrenalin, 5 parts; tartaric acid, 5 parts; sodium chloride, 90 parts; 0.15 of a grain of this powder added to ten drops of water. The author recommends applications of the solution thus made every two days. Not only cancers of the larynx and tongue were benefited by the treatment, but also those of other regions, including the breast.—*Revue francaise de médecine et de chirurgie*, 1904, No. 31, p. 741.

The Bloodless Treatment of Furunculosis.—DR. SEMBRITZSKI has employed the following treatment in a series of cases, some of which were very severe, with excellent results: First the skin is cleansed with ether and shaved, then the entire inflammatory area is covered with a wet gauze dressing of absolute alcohol and hermetically sealed. As an adjunct to the treatment the patient is given beer yeast or some one of its derivatives, such as "zyma." The dose of the former is a tablespoonful three times a day; of the latter a teaspoonful. These are given in water or beer. In twenty-four hours the dressing is removed, and in favorable cases the swelling, redness, and œdema will be found much diminished. In such cases the treatment is continued, but unless an amelioration takes place within the first twenty-four hours the use of the knife is indicated.—*Wiener klinische therapeutische Wochenschrift*, 1904, No. 29, p. 781.

The Treatment of Insomnia of Cardiac Origin.—L. FEILCHENFELD advises in the insomnia which is present in certain cases the use of the following heart tonic: One-fifth of a grain of powdered digitalis leaves are mixed with one-thirtieth of a grain of morphine hydrochlorate, and the powder is taken at bedtime. The author believes that this insomnia, which is characterized by a slumber of an hour or two early in the night, followed by wakefulness accompanied by weariness, oppression, and palpitation, is due to cardiac insufficiency and passive dilatation. The small dosage recommended is usually sufficient to give relief, and no unpleasant after-effects, such as may follow larger doses, are noted.—*Journal de médecine de Paris*, 1904, No. 28, p. 280.

The Treatment of Aneurysms of the Aorta by Injection of Gelatin.—Dr. Lancereaux has injected the following solution subcutaneously, with the best of results: distilled water, 1000; sodium chloride, 7; gelatin, 25 parts. The gelatin must be of the best quality, and the solution should be sterilized several times under pressure at a temperature of 110° to 115° C. Each injection consists of about six ounces, and the treatment is repeated every four or five days. The procedure causes a cessation of the pain and a hardening and shrinkage of the sac. The patient may return to work, provided he avoid exertion. The author concludes thus: The method is not dangerous, provided the gelatin be pure and thoroughly sterilized. The injections favor the coagulability of the blood in the aneurysmal sac, and thus tend to cure. Tetanus or other complications are always due to poor gelatin or lack of sterilization.—*Gazette médicale de Paris*, 1904, No. 28, p. 328.

The Alkalies in Diabetic Coma.—Dr. Lenne reports a case which after having been affected for two years with diabetes suffered a typical attack of coma. In addition to alcoholic stimulation a solution of a mixture of two and a quarter ounces of sodium bicarbonate and one ounce of magnesium carbonate was administered. The patient gradually recovered until in three days all signs of the coma had disappeared. Shortly afterward he suffered a second attack, which was also controlled, but larger quantities of the alkalies were given. This case is in direct contrast to three similar cases in which the alkaline treatment had no effect.—*Deutsche Aerzte-zeitung*, 1904, No. 8, p. 180.

Validol and Seasickness.—Dr. K. Koepke has found that in the early stages of seasickness 10 to 15 drops of validol upon a lump of sugar, with advice to lie down for half an hour, at the end of which a glass of wine and a biscuit may be taken, is sufficient to prevent further symptoms. In more marked cases which have been prostrated by a day's *mal de mer* the patient should be put to bed and given validol on sugar as above. After half an hour the yolks of two eggs, mixed with sherry and ice, are given by teaspoonful; without exception this will be found to be well borne. After this procedure has been carried out several times in the course of the day, the patient may be allowed light diet. —*Therapeutische Monatshefte*, 1904, No. 6, p. 296.

The Action of Strophanthus.—Dr. G. Günther, after an examination into the action of this drug upon horses, sheep, dogs, and other smaller animals, has arrived at the following conclusions: 1. Strophanthus is purely a heart muscle poison in mammals, and produces death by causing stoppage of the heart in diastole. 2. In poisonous doses it causes characteristic changes in the pulse and blood pressure, different from those caused by digitalis through the absence of stimulation of the vagus. 3. Strophanthus is a vasoconstrictor, but is less active in this regard than digitalis; this action is, however, not marked in overdosage. 4. Strophanthus possesses a specific action upon the vomiting centre. 5. Of antidotes the chief place is occupied by artificial respiration. Excitants are without influence, as they are in poisoning by morphine and chloral hydrate.—*Therapeutische Monatshefte*, 1904, No. 6, p. 285.

OBSTETRICS.

UNDER THE CHARGE OF

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Ovarian Tumors during Pregnancy and the Puerperal Period.—Lea (*Journal of Obstetrics and Gynecology of the British Empire*, August, 1904) contributes a paper upon this subject, reporting four cases. He quotes the frequency of ovarian tumors during pregnancy as 5 in 17,832; Flaischlen, 2 in 1300; Lohlein, 1 in 1738; Tarnier, concluding that 1 in 2500 is the average frequency. At St. Bartholomew's Hospital Cripps had 2 cases in 182 patients with ovarian tumors.

Dermoids are more frequent in the proportion of 4 out of 7, about 25 per cent. Torsion of the pedicle occurs in from 6 to 12 per cent. during pregnancy, and during the puerperal period in about 22 per cent. Rupture is found in from 3 to 4 per cent. In Lea's 4 cases torsion occurred in 2; necrosis of the cyst followed by rupture and peritonitis during the last month of pregnancy occurred in 1, and impaction of a dermoid cyst in 1 case. In 12.5 per cent. Williams found that pregnancy was interrupted by the presence of the tumor.

The prognosis, so far as death is concerned, in these cases is fairly good. In 461 cases Williams found only 1 of sudden death from rupture, and 6 in which the tumor ruptured during pregnancy, when the patients died after labor. Labor and the puerperal period greatly increase the danger and bring the total mortality up to 25 per cent.

Regarding treatment, patients without operation show a mortality of 21 per cent.

Lea describes the case of a woman, aged forty-two years, in her sixth pregnancy at eight and one-half months. She had been in bed for six weeks, with severe abdominal pain and nausea. She had slight jaundice. Her temperature was from 100° to 101°; her pulse 120 and weak. The pelvis was occupied by a fixed tumor preventing the engagement of the head. Operation was done through the vagina, and a necrotic cyst adherent to the anterior wall of the rectum was removed in fragments. The pedicle was recognized and ligatured. The patient collapsed, but rallied with saline transfusion. Labor was spontaneous twenty-four hours later, a living child being born. The patient recovered, but died six months afterward from myelitis.

When an ovarian tumor obstructs labor its removal is imperative before an effort is made to deliver the child. In fifteen cases of ovariotomy during labor all recovered. In cases otherwise treated the mortality rose to nearly 50 per cent.

During the puerperal period ovarian tumors are dangerous through torsion of the pedicle, peritonitis, and suppuration, and necrosis in the cyst. Cysts often become infected when general septicæmia develops.

Lea reports the case of a woman delivered spontaneously of a living child who four days later had all the symptoms of puerperal infection. A hard mass of exudate in Douglas' pouch was found pushing the uterus forward. In performing posterior vaginal section a large quantity of pus was evacuated and a drainage tube inserted. The patient recovered.

Five years later she was again delivered of a living child in natural labor. On the third day infection developed, and a tumor the size of a large orange was found behind the uterus. On posterior vaginal section the tumor was readily exposed, adhesions broken up, the pedicle tied with silk, and the tumor removed. It was a dermoid of the left ovary. The pelvic cavity was flushed and packed with gauze, the patient making a good recovery. No organisms were discovered in the tumor or fluid. Without operation the post-partum mortality of these cases is about 28 per cent. In operation during the puerperal period a mortality of 14 per cent. is reported.

Post-partum ovariotomy is often remarkably successful. McKerron collected 92 cases with 79 recoveries—a mortality of 14 per cent. In 2 of these cases gangrene was present, and in 2 cases the tumor was malignant. From twenty-four to forty-eight hours after delivery is the most favorable time for this operation.

The Automatic Action of the Uterus and the Effect of Drugs upon Uterine Contractions.—In the *Archiv für Gynäkologie*, 1904, Band lxxiii., Heft 2, KURDINOWSKY reports the results of experiments made in the pharmaceutical laboratory of Professor Krawkow at St. Petersburg. Under ether the uterus of a rabbit was removed and placed in a neutral fluid at a temperature of 38° C. The whole was placed in a warm, moist chamber. Ludwig's kymographion was used to study the uterine contractions.

Immediately after removal from the body the uterus seemed relaxed. In about half an hour automatic contractions began earlier in the pregnant than in the non-pregnant uterus. The tissues of the uterus and vagina contracted separately, the uterus after an hour becoming apparently fatigued, and after an hour's rest again contracting. The uterine muscles began to act at the cornu, peristalsis continuing through the uterus to the vagina. To continue the experiment the temperature at which the uterus was kept was reduced three or four degrees, when it was found that contractions of the uterus persisted and could be excited in one case forty-nine hours and forty minutes after the removal of the womb from the body of the animal.

When this action of the uterus was studied it was observed that the muscle began its work at each cornu in alternation. The effect of this would be to force a body like the ovum downward into the uterine cavity. When the uterus had accomplished this the broad ligaments contracted in a direction transverse to the long axis of the womb. This action lasted but for a certain time, and when the ovum reached the vagina the broad ligaments ceased to contract. Each half of the uterus and broad ligament acted independently of the other, but in alternation. This action was entirely automatic and absolutely without the influence of the central nervous system.

In experimenting upon the pregnant uterus it was found that if blood were entirely washed out of the womb by injecting salt solution the ovum

died as soon as the womb became anæmic. The contraction of the uterine muscle, however, continued for fifty minutes after the uterus had been deprived of blood. For thirty-five minutes of this time the uterus was without the body of the mother. It was found that hot and cold stimulated uterine contraction to an equal degree. Variations in temperature seemed more stimulating than either heat or cold. At the usual termination of pregnancy the uterus is in a condition automatically to expel the fœtus.

In experimenting with drugs upon uterine contraction hydrastis, canadensis, ergot, chloral hydrate, and adrenalin were employed.

Hydrastin produced tetanic contraction of the uterine muscle, but did not affect the bloodvessels of the uterus. Ergot and its preparations had the same effect that hydrastin had on the uterus. Narcotics, as chloral hydrate and alcohol, had little effect upon the uterine muscle. When given to the point of narcosis their action became apparent.

Adrenalin in very weak solution produced marked effect upon the womb. In 1:10,000,000 it considerably increased the strength of uterine contractions. If pushed to any extent it produced well-marked tetanus of the uterine muscle. It produced an extraordinary vigorous effect upon the vessels of the uterus, causing them to contract. Experiments upon living animals confirmed those made upon the exsected uterus.

The Prevention of Morbidity during the Puerperal Period.—KROEMER (*Monatsschrift für Geburtshülfe und Gynäkologie*, 1904, Band xx., Heft 2) draws attention to the value of taking all possible precautions to prevent complications during the puerperal period. During 1893, in the Marburg Clinic, the percentage of morbidity was 38.7. Severe infections occurred in 1.07 per cent., while the mortality from septic infection was nothing.

In preventing complications during the puerperal period the introduction of rubber gloves had unexpectedly been without result. It must be remembered that even in healthy patients the vagina contains germs capable under some conditions of becoming actively pathogenic. For this reason, although with rubber gloves, the hand may be practically sterile, its use cannot entirely remove causes of puerperal infection. The effort to destroy these germs by vaginal douching and cleansing exposes the patient to such danger of uterine infection as to be undesirable. Furthermore, in from 20 to 22 per cent. of pregnant patients the secretion of the vagina is pathological in character.

As regards labor itself, patients admitted to the clinic with fever are confined in rooms set apart for such cases. If a patient, already in the house, develops fever during labor the confinement-room is immediately disinfected. Every precaution is taken to at once stamp out the infection. Except in infected and suspicious cases the use of gloves is not insisted upon, nor considered of practical value.

In the lying-in ward all who have to do with the care of patients are required to use gloves. Douches are given during the first two or three days, and nurses use sterilized forceps to handle cotton sponges and other dressings. Every precaution is taken to avoid the spread of contagion from one patient to another. Cases developing fever are at once isolated.

When patients are received and examined before labor the entire body is thoroughly examined, and in addition the character of the

vaginal secretion is ascertained. When this is pathological and abnormal a vaginal douche of 1:2000 bichloride is used. If gonorrhœa is present rheol, followed by a douche of lysol ½ per cent., are used. All pregnant patients are examined twice weekly, and the genital region thoroughly explored to detect ulceration or signs of infection. The legs are examined for varicose veins or ulcers. Patients are obliged to bathe daily, and strict precautions are taken about linen.

The breasts are inspected, the nipples thoroughly washed, and alcohol used with glycerin and boric acid for local antisepsis. The danger of erysipelas arising from excoriation of the nipple is considered great, and every precaution taken to avoid this accident. Areas of distention caused by the accumulation of milk do not produce serious consequences.

In choosing antiseptics for douches care should be taken to employ dilute solutions to avoid irritation and injury. With such precautions the morbidity percentage of several clinics shows about 30 in operative cases and less than 15 in cases of spontaneous parturition. It is considered of especial importance that pregnant patients be kept under complete and absolute control and that the reduction of complications in labor and the puerperal state will depend upon the thoroughness and perfection of this precaution.

GYNECOLOGY.

UNDER THE CHARGE OF

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Leukocytosis in Gynecology.—PANKOW (*Zentralblatt für Gynäkologie*, 1904, No. 11) states that in all cases in which there is a persistent leukocytosis of over 10,000 (without any other satisfactory explanation) the presence of pus in the pelvis should be inferred. It cannot always be excluded with a lower count, since there may be either a tuberculous focus, or the abscess may be surrounded with such dense adhesions that no absorption occurs. Hæmatoceles, so long as they do not suppurate, are unaccompanied by leukocytosis. A post-hemorrhagic leukocytosis may, however, follow rupture of a tubal gestation.

Torsion of the pedicle of an ovarian cyst is rapidly followed by an increase in the white cells, possibly due to the accompanying irritation of the peritoneum, but in acute peritonitis this may be absent, as the power of reaction of the system is increased. Leukocytosis, from 11,000 to 13,000, is a more reliable indication of pus than the temperature curve (?).

In cases of cancer no inference can be drawn from leukocytosis with regard to the locality, character, or extent of the disease. Even when

necrosis is present (unless there is actual pyometra) there is no increase in the white cells, probably because the sloughs are rapidly cast off and there is little absorption. No increase is observed when the lymph nodes are affected. Among the cases of fibromyoma which were studied with especial reference to leukocytosis, this was noted in only a single instance as a post-hemorrhagic phenomenon.

After the evacuation of abscesses by colpotomy a diminution in the number of leukocytes was promptly noted, unless pus-foci were not thoroughly drained. After abdominal section in pus cases there was at first a marked increase, which gradually declined to the normal if the case progressed smoothly. Imperfect drainage, mural abscess, etc., caused a fresh increase in the number of leukocytes. Even in aseptic cases the same degree of leukocytosis was noted on the first day after operation, although there might be no accompanying elevation of temperature. The writer explains this phenomenon on the theory that in every operation, no matter how aseptic, a certain amount of infectious material is introduced into the wound, the absorption of which into the circulation produces a marked irritation of the leukocytes.

Further observations during normal pregnancy and labor led the writer to the following conclusions: Leukocytosis is seldom found at the end of pregnancy. There is an increase during labor, most marked if the pains are severe and the labor prolonged. The maximum is reached an hour or two after the completion of the third stage. There is a gradual decline to normal on the fourth day.

The Uterus in Ectopic Gestation.—CAZEAUX (*Revue prat. d'obstétrique et de pediatrie*, November, 1903) finds that in ectopic gestation the uterine mucosa is intensely congested and hypertrophied, interstitial hemorrhages being frequent. This explains why the passage of the curette usually causes bleeding. The pseudomembrane is rarely expelled in the shape of casts of the uterine cavity, the shreds being more often overlooked in the accompanying blood. This usually occurs early in the pregnancy, and does not by any means imply the death of the product of conception, but it is usually indicative of rupture or tubal abortion.

Cancerous Degeneration of the Cervix after Supravaginal Amputation.—RICHELOT (*Annales de gyn. et d'obstétrique*, December, 1903) concludes a paper on this subject with a positive expression of his preference for pan-hysterectomy in cases of fibroid where a radical operation is indicated.

He also believes that fibromyomata are only relatively benign. While, of course, it is not always necessary to remove them, he has seen too many patients with these neoplasms succumb to phlebitis or visceral disease, while under observation, to regard them as entirely innocent. While sarcomatous degeneration is rare, he does believe that cancer frequently develops in fibroid uteri in consequence of the accompanying endometritis. He cites two cases from his own practice and ten from the literature in which the cervix became cancerous after supravaginal amputation.

Obliteration of the Tubes.—RONSSE (*Annales de gyn. et d'obstétrique*, December, 1903), from experiments on animals, concludes that the

lumen of the tube can be completely obliterated by section between ligatures. He obtained the same results in six operations upon the human subject, and prefers this method to the more complicated ones that have been proposed. It cannot be denied that there is always a certain amount of risk of subsequent dilatation of the tube, so that the operation should not be resorted to unless it is clearly indicated, as after Cæsarean section, in tuberculous and cardiac cases, etc.

The writer criticises the operation proposed by Kehrer as liable to abuse and not free from danger.

Histology of Chronic Oophoritis.—PINTO (*Zentralblatt für Gynäkologie*, No. 23, 1904), after reviewing the various conflicting views with regard to the inflammatory nature of this affection, states the conclusions to which his own histological studies have led him. He objects to the division into interstitial and parenchymatous oöphoritis, because it is hardly possible to separate the two varieties, even in the acute form. Since in some cases the cortical layer is first affected by contiguity, and in others the deeper portions of the ovary are primarily infected through the medium of the bloodvessels and lymphvessels, he suggests the terms "cortical" and "diffuse" oöphoritis. In the former the albuginea is thickened, partly from adhesions, the ovary being usually of nearly normal size; and, if the case is not of long standing, round-cell infiltration will be observed in the neighborhood of the vessels. Later portions of the stroma will be converted into firm fibrous tissue, containing islands of normal tissue with a few primary follicles. In the latter atrophy of the nuclei and degeneration of the protoplasm will be seen, especially in the more superficial follicles. They are not increased in number, and in some the epithelium and ovum may be practically unchanged.

In the diffuse variety the ovary is cirrhotic, with hyperplasia of the stroma and a diminution of the number of follicles, which, however, are normal or exhibit a tendency to atresia or cystic degeneration. The vessels show hyaline degeneration of the middle layer of the intima, obliterating endarteritis, or perivascular sclerosis. Round-cell infiltration is rare, but hemorrhagic foci are seen, both in the stroma and in the follicles. The appearances correspond to those usually described as interstitial oöphoritis, which may result in general sclerosis. The writer dissents from the opinions expressed by Martin and Orthmann, that cystic degeneration of the ovary is necessarily the result of a chronic inflammatory process in or around the ovary. As regards etiology, he believes that infection (gonorrhœal or septic) plays the principal role, though chronic congestion from any cause may act as a predisposing factor.

Changes in the Uterus in Ectopic Gestation.—CAZEAUX (*Revue prat. d'obstét. et de pediatrie*, 1903, No. 11) says that in connection with tubal pregnancy the uterus is gradually pushed forward and to one side. If many adhesions are present it may be impossible to map out the body of the organ. The cervix remains softened, he affirms, as long as the product of conception is living, though this softening is not so marked as in normal pregnancy. The same applies to the corpus uteri, which becomes notably smaller and harder after the death of the fœtus. The writer differs from some authorities in holding that pseudomembrane

may be cast off from the uterine cavity at any time during the extra-uterine pregnancy and that it does not necessarily point to the death of the fœtus, though it is often significant of hemorrhage into the tube or tubal abortion.

Adenoma of the Fallopian Tube.—NADORY (*Zentralblatt für Gynäkologie*, 1904, No. 23) reports the following cases:

Case I. A II-para, aged twenty-eight years, had suffered for three weeks with abdominal pains and a bloody discharge. A diagnosis of incomplete abortion with enlargement of the left tube was made, and the uterus was curetted. As the tube did not diminish in size under treatment and the patient continued to suffer great pain, laparotomy was performed. A pus-tube was removed, which was thought to be tuberculous, but on microscopic examination it proved to be a typical adenoma.

Case II. The patient, aged twenty-nine years, had been sterile for eight years, and was sent to the clinic as a case of probable ruptured ectopic gestation. On opening the abdomen general adhesions were found, the affected tube was removed, and showed at its isthmus a myoadenoma, such as was originally described by Recklinghausen, the mucosa being intact. The writer regards this obstruction of the lumen as the cause of the tubal pregnancy.

Extirpation of the Spleen.—CASATI (*Zentralblatt für Gynäkologie*, 1904, No. 23) reports five cases, in three of which the spleen was enlarged from malaria, once from cancer, while there was one case of idiopathic enlargement. The patients all recovered, those with the malarial spleens being cured. The writer does not advise splenectomy for this cause unless the tumor has reached a very large size. He makes a medium incision and uses a Mikulicz drain if extensive adhesions have been separated.

Metrorrhagia in Syphilis.—OZENNE (*Soc. médico-chirurg. de Paris; La gynécologie*, June, 1904) calls attention to the fact that this symptom is generally observed during the tertiary stage, but that it may appear during the secondary, and may be of diagnostic value in the absence of local evidences of infection. He reports two cases which yielded to mercurial treatment after all local measures had failed.

Drainage after Laparotomy.—SCHWEIZER (Inaugural Dis.; abstract in *Zentralblatt für Gynäkologie*, 1904, No. 23), from an analysis of a series of cases at the Zurich clinic in which drainage was employed by means of gauze wrung out in sterile water, or a solution of salicylic acid, and brought through the lower angle of the wound, arrives at these conclusions: In cases in which there is doubt as to the sterility of the pus in a case of tubal or ovarian abscess, and when a perforation of the intestine has been sutured, the gauze drain or tampon should always be used, since in this way many fatal results can be averted.

[We have cited his paper in order to emphasize the expression "or tampon," since the writer, by stating the fact that gauze acts as a tampon, as well as a drain, involuntarily admits the weak point in his argument. He evidently has not followed the trend of modern surgical technique, which is that so-called gauze-drain (especially when it is

introduceed through a small abdominal wound) more often prevents than favors the escape of fluid. In those exceptional cases in which gauze-tamponade of the pelvis is employed it is generally admitted that the vaginal route is the natural one for drainage.—H. C. C.]

OTOLOGY.

UNDER THE CHARGE OF

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Injuries of the Drumhead from Indirect Violence.—W. BOEHM (*Monatsschrift für Ohrenheilkunde*, March, 1904) states that injuries of the drumhead, in the form of a solution of its continuity, are more frequently the result of indirect violence than of puncture by foreign bodies, destruction by chemical action, or perforation as a consequence of extreme temperature limits locally applied. This is in accordance with the fact that even a very moderate degree of air pressure applied in such fashion as to force the drumhead to make a major excursion within a limited period of time is sufficient to ensure its rupture.

It is not the forceful box upon the ear which ruptures the drumhead so often as the less forcible blow with the flat of the hand covering the whole auricle, and, for a moment, making an air-tight closure of the canal; and it is interesting to note that the clinical records of cases of injury to the ear from this cause show the majority of them to have occurred on the left side, a natural result of the preponderance of right-handed blows.

That the condition of the drumhead itself should exert an influence upon the extent and location of the injury resulting from a sudden excursion under the influence of air pressure is evident, as illustrated in cases of partial atrophy of the drumhead, of cicatricial adhesions, or of the weighting of certain portions by the presence of calcareous deposits.

That detonation at short range may cause a rupture of one or both drumheads is not infrequently observed in artillery service, and the same is true in cases of dynamite blasters, in divers, and in caisson workers. Injury of the same character may also be caused under conditions of exhibition of pressure from within outward, by sudden compression of the air within the middle ear, as in violent coughing, sneezing, and vomiting, or by the use of the Politzer air douche and, contrariwise, by the sudden exhaustion of the air in the external canal, as in a case reported by Huettig, where a kiss upon the ear resulted in a rupture of the drumhead.

There is still another group of injuries to the drumhead, those resulting from cranial concussion, which has not, as yet, been sufficiently

classified, and which includes not only cases of simple contrecoup, but of double-sided injury as well.

Of these latter cases the author has found but one record in reviewing the literature of the subject, the case reported by Stankowsky, of a young man who had perforation of both drumheads as the result of a heavy blow upon the left ear, in which ear there was also a labyrinth concussion as further evidence of the force of the impact.

The exact mechanism of the injury by contrecoup is not yet adequately determined, and it is possible that help for solution of the general question may be reached through carefully collated records of injury to the drumhead and such observations as have been attempted by Corradi, who finds that the majority of solutions of continuity resulting from air pressure occur in that portion of the drumhead which is both the thinnest and the most movable, namely, the central portion, while the injuries as the result of contrecoup are, as a rule, longitudinal ruptures starting from some point at the periphery of the drumhead and running wholly or partly across it, a condition which he regards as consonant with the proposition that in the transference of motion from a medium of one density to that of another of different density, the point of transference is that at which the solution of continuity is most likely to occur.

With this opinion of Corradi the author does not, however, agree, holding that ruptures of this type are not the result of irradiation, and the transference of the force exerted to a weaker medium, but are exhibitions of the true contrecoup reaction.

This view is based upon the experiments of Bergmann and Messerer upon the elasticity of the bones of the skull, considered as parts of an elastic globe, with susceptibility to "bursting fracture" primarily at points at right angles to the line of compression, and, secondarily, where there is a simple blow without counteracting pressure, midway between the point of impact and the right angle above mentioned; in other words, the author holds that ruptures of the drumhead consequent upon blows upon the skull other than those directly applied to the affected ear are the result neither of compression nor of irradiation, in the sense of Corradi's theory, but of a true "bursting fracture" incident to the natural elasticity of the skull which makes the phenomenon of contrecoup possible.

The Surgical Treatment of Peripheral Paralysis of the Facial Nerve.—G. ALEXANDER (*Archiv für Ohrenheilkunde*, Band lxii, Heft 1–2.) reports a case of grafting of the facial nerve to the hypoglossal. The patient presented complete left-sided paralysis of six years' duration, as the result of chronic suppurative middle-ear disease. The radical operation was performed and the labyrinth found diseased and filled with polypoid granulations. The facial and its canal were completely destroyed by the disease. Examination of the region supplied by the left facial showed the orbicularis oris somewhat contractile, but complete paralysis of the other face muscles and of the platysma. There was no disturbance of the taste sensation on the tongue, and there was a normal condition of the soft palate. Galvanic and faradic stimulation of the muscles by way of the nerve gave no reaction. However, direct faradic stimulation showed a slight reaction of the orbicularis oris of the under lip and direct galvanic stimulation showed evident reaction

of the orbicularis of the upper and under lip. The grafting of the facial and hypoglossal was accomplished by exposing the hypoglossal in the angle made by the two bellies of the digastric muscle, dissecting out the facial trunk where it emerges from the stylomastoid foramen, cutting this off obliquely (a small bit of the nerve taken from this end and subsequently examined microscopically showed complete degeneration of the nerve fibres), and then suturing this end to the side of the hypoglossal nerve after denuding a small area on the side of this nerve of its neurilemma.

Six weeks after the grafting, the condition was as follows: Indirect faradic stimulation gave no reaction. Indirect galvanic stimulation (the electrode was applied over the scar of operation) showed slight reaction of the orbicularis oculi and the orbicularis oris of the upper and under lip, also of the depressor anguli oris. Direct faradic stimulation showed reaction in the orbicularis oris of the under lip and direct galvanic stimulation showed slight reaction in the same muscles which responded to indirect galvanic stimulation.

Alexander reviews the cases of suture of the facial to the spinal accessory reported by Ballance and that of Faure, where the facial peripheral end was sutured to the proximal end of the completely separated hypoglossal. A case of Körte of grafting similar to Alexander's own case and a case of facial-spinal accessory suture by Glück are briefly reviewed. Three methods have been employed:

1. Grafting of the several facials to the spinal accessory.
2. End-to-end suture of the peripheral end of the severed facial to the proximal end of the severed hypoglossal.
3. Grafting of the severed facial-peripheral end to the hypoglossal.

Of these methods Alexander believes the third most desirable because, in the first method, there are associated movements of the shoulder; and in the second method, although an end-to-end suture gives the best chance for regeneration of the nerve fibres, a permanent one-sided paralysis of the tongue and a consequent atrophy of the paralyzed side results. In no case thus far observed has a complete restoration of the function of the facial resulted.

As to the time after the onset of the facial paralysis at which the grafting should be undertaken, Alexander believes:

1. The grafting of the facial should not be undertaken before six months after the time of the appearance of the paralysis, and during this time, massage and electric treatment should have been tried.
2. After six months all cases should be operated in which (*a*) no voluntary contraction has become possible; (*b*) in which no faradic excitability is present; (*c*) in which the galvanic excitability of the nerve, in spite of treatment, decreases.
3. In cases of more than six months' duration a favorable result of grafting can be expected only when there remains some direct galvanic excitability.
4. If, in old cases, there is prompt galvanic reaction or there is a remnant of faradic excitability, some conservative treatment for a time is indicated.

Middle-ear Disease in its Relation to Metastatic Abscess of the Liver and Other Viscera.—S. MACUEN SMITH (*Archives of Otology*, April, 1904) reports three interesting cases of metastatic abscess of the

liver of otitic origin. While almost all of the cases of abscess of the liver are due to such causes as dysentery, appendicitis, gastric ulcer, etc., he believes that the so-called cases of "spontaneous abscess" of the liver may be due to primary foci in the ear.

The first case is that of a man, aged forty-two years, who had suppurative disease of the middle ear for twenty-seven years. For the past twenty years he suffered no inconvenience beyond a slight discharge, with the exception that he has had increase in the amount at intervals for three years. The writer saw the patient during one of these attacks. After a chill the temperature registered 105.2°, followed by profuse sweating. He complained of severe pain in the right shoulder and neck, and a moderate yellow tinge of the skin was noticed. The aural examination showed a complete destruction of the membrana tympani, malleus, and incus. Pus was present in the canal, and was very offensive. Examination of the mastoid process and adjacent parts was negative. The severity of the above symptoms increased, and a week before death the liver was found to be enlarged. There was also pain in the hypochondrium. The temperature was pyæmic in character, and just before death registered 106°.

Antistreptococcic serum was used, but with no beneficial effect. At the autopsy all the organs were negative, except the liver, which was a mass of miliary abscesses.

A second case is reported similar to the first, with the exception that the duration was of eight years and the discharge was not so offensive. The autopsy revealed multiple abscesses in both the liver and lungs.

The last case is that of a man, aged twenty years, who had suppurative disease of the middle ear since an attack of measles when he was four years of age.

Previous to the present attack the ear had been dry for six months. Two days after the discharge began he complained of severe and constant pain in the vicinity of the right ear. Temperature at this time was 98°. Two days later the temperature rose and the pain increased. The mastoid was found to be tender, and an operation was performed at once. The bone was necrosed, and the cells filled with pus and granulations. No evidence of sinus involvement. Two days later the temperature rose to 104.4°, the dressing was removed, and the wound found to be very offensive; 30,000 units of anti-streptococcic serum were given. The next day a second operation was performed, the lateral sinus being opened and found to be thrombosed. This was curretted, and the temperature fell to 97°. Seven days after the second operation the patient had a chill, and the temperature rose to 105°. Chills occurred at different intervals for a week, when tenderness over the jugular vein being noticed, an incision was made and the vein found to be healthy. The patient complained of pain in the right shoulder and neck, and the liver was tender. Death occurred about a month after the onset of the disease. The autopsy revealed a mottled appearance of the brain at the seat of the mastoid operation. The lung, liver, and spleen showed many miliary abscesses. The heart and kidneys were negative.

The writer summarizes the most important features as follows:

1. Acute exacerbation of a chronic process—an early symptom.
2. The severe pain in the right shoulder and neck beginning at the same time as the hepatic suppuration.

3. Pain and tenderness of the right hypochondrium, indicating hepatic disease.

4. Repeated chills, pyrexia, and diaphoresis, indicating a septic process likely to prove fatal.

5. The marked accessions and remissions of temperature, typical of hepatic abscess.

6. While jaundice was a constant feature it was of little diagnostic value.

7. The conclusion drawn is that metastatic abscess of the viscera may be caused by suppurative disease of the middle ear.

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES.

UNDER THE CHARGE OF

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Pelliculous Sore-throats.—The profession has long been aware of the existence of various pelliculous sore-throats which were not diphtheria, and these have caused considerable confusing controversy. Bacteriologic investigation is gradually differentiating these affections. One variety, now known as Vincent's angina, has been the subject of careful study during the latter half of the present decennial. An excellent summary of what is known about this form of pelliculous sore-throat has been written by DR. RANDLE C. ROSENBERGER (*American Medicine*, July 23, 1904), who reports in addition two cases of Vincent's angina and one of noma in which the organisms characteristic of the former affection were found. DR. THOMAS H. HARRIS (*ibid.*), of New York, reports two cases, and discusses a differential diagnosis under the caption "Primary Ulceration of the Tonsil," together with some remarks on tonsillar ulceration in general. DR. G. C. CRANDALL, of St. Louis (*Journal of the American Medical Association*, July 23, 1904), reported a case of Vincent's angina and stomatitis at the late meeting of the Section on Stomatology of the American Medical Association. The patient was a medical student in fairly good health, with throat, mouth, and teeth in good order. The affection began in the tonsil and palate, and extended to the gums in consequence of cleansing of the teeth by a dentist on the fourth day of the disease.

Adrenalin in Malignant Disease of the Throat.—DR. AIMAR RAOULT, of Nancy, reports (*Revue hebdomadaire de laryngologie, d'otologie et de rhinologie*, Juillet, 1904) a case of a man, aged sixty-eight years, whose right tonsil had in great part been removed by excision. Subsequent developments evincing malignancy, treatment with applications of adrenalin hydrochlorate, 1:1000, were instituted. While almost complete elimination of the tumor was eventually accomplished under the action of the adrenalin, serious œdema of the entire region had been

produced, including the anterior and posterior pillars, the uvula, and a great portion of the soft palate, so that the applications of adrenalin had to be abandoned temporarily. The patient was lost sight of, but was found to have died a few weeks after cessation of the treatment.

Primary Tuberculous Growth of the Nasal Septum.—At a recent meeting of the Laryngological Society of London (*Journal of Laryngology, Rhinology and Otology*, August, 1904) DR. SMURTHWAITE showed a microscopic slide from a growth removed from the anterior portion of the cartilaginous septum on the right side of a woman, aged thirty-five years. The tumor was bluish-white and very uneven in surface. It was readily removed with a Volkmann spoon. The cartilage was then scraped bare, and the surface rubbed thoroughly with lactic acid solution, 75 per cent. Three months after treatment there had been no signs of recurrence.

As shown in the slide, the nature of the growth was undoubtedly tuberculous, for, though no bacilli were found, a number of giant-cells were seen to be present.

Carcinoma of the Nose.—At the late meeting of the Laryngological Society of London (*Journal of Laryngology, Rhinology and Otology*, August, 1904) DR. FELIX SEMON showed a case of papilliferous columnar-celled carcinoma of the nose in a young man, aged twenty-four years. The patient had been sent to Sir Felix in consequence of recurrences after removal of a cauliflower growth from the region of the left meatus, and which apparently sprung from the septum high up from beneath the cribriform plate. Examination of specimens of the original growth showing it to be a papilliferous columnar-celled carcinoma, by advice of Sir Felix Semon, Sir Victor Horsley performed a very radical operation, as follows: He first ligated the external carotid, then, after plugging the rhinopharyngeal cavity, he did a Rouge operation, and removed the greater part of the bony septum, the left middle turbinate, and the ethmoid on the left side up to the cribriform plate. The operation occupied nearly two hours, but so far as could be judged succeeded in completely removing the growth with a healthy area around it. The patient made an uninterrupted recovery, and there had been no recurrence.

Cancer of the Ethmoid.—DR. MARCEL LERMOYEZ (*Annales des maladies de l'oreille, du larynx*, etc., Juillet, 1904) reports a case of cancer of the ethmoid in a man, aged forty-five years, which he removed after external exposure by resecting not only the nasal process of the upper jaw, but also the nasal bone proper, except its anterior border and the nasal spine of the corresponding frontal bone. This enabled him to expose the ethmoid frontally, instead of obliquely, as when access is made by way of the orbit. The growth, which on inspection through the natural passages was supposed to be the size of a nut, was found to be larger than an egg. It occupied the entire ethmoid, extending in front of the *agger nasi*, where it would have been totally impossible to reach it by way of the natural passages. The tumor was easily removed with the large ethmoidal forceps of Luc, which permitted its extraction in large fragments without notable hemorrhage. As there were no prolongations, its total ablation was practicable. The patient made a

good recovery, and left the hospital on the seventeenth day able to resume his work. Three months later, when the patient was shown to the French Society of Otology and Laryngology, June 15th, the local condition was perfect, a cicatrix barely visible indicating the line of incision through the soft parts.

The Utilization of the Lower Lip in Rhinoplasty: A New Procedure.—Dr. Carl Beck, of New York, exhibited to the Surgical Section of the Academy of Medicine (*Medical News*, March 5, 1904) a woman, aged fifty-five years, in whom he utilized portions of the lower lip and chin for replacing lost tissues of the left side of the nose. The patient had been operated upon about three years previously with flaps taken from the forehead and cheek, which, though apparently successful at the time they were made, had disappeared in a recurrence by the end of a year. In the absence of available tissue in the vicinity of the area, Dr. Beck operated by taking a large flap from the left arm. Eighteen months later another recurrence was followed, with more extensive destruction; the mucous membrane of the left nasal passage being completely exposed. The defect was covered by a triangular flap from the lower lip. The details of the procedure are illustrated by a diagram and three photographs. The advantage of this procedure seemed to the operator to lie especially in the possibility of supplying a mucous membrane throughout the whole area of the defect so as to restore normal conditions as far as practicable.

Spontaneous Disappearance of an Obscure Ulceration in the Larynx.—At the late meeting of the Laryngological Society of London (*Journal of Laryngology, Rhinology and Otology*, August, 1904) Sir Felix Semon showed a case of obscure ulceration of the left vocal cord of nearly a year and a half standing in a gentleman, aged about sixty years, which had since disappeared spontaneously. The case had been shown as an extremely obscure one, and fully described at the December meeting, 1903, when the discussion revealed considerable differences of opinion. Since then the patient, after an attack of influenza, became extremely ill, so that his life was despaired of for a considerable time. He was again seen, after an interval of several months, at the beginning of May, when it was found that the chronic and troublesome ulceration, which had existed for so long a time, had spontaneously and completely disappeared, so that there only remained slight congestion and relaxation of the left vocal cord. The question as to the nature of the chronic ulceration is thus more obscure than ever.

[In the compiler's experience epithelioma, syphiloma, and even tuberculosis of the throat have been known to undergo spontaneous retrogression during severe attacks of influenza and likewise of erysipelas.]

Tumor of the Larynx: Successful Treatment by Roentgen Rays in Neoplasm of the Larynx.—Drs. A. Beclere and Paul Viollet (*Revue hebd. de laryngologie, d'otologie et de rhinologie*, Juillet 9, 1904) report an extensive case of a laryngeal tumor supposedly malignant, apparently cured—temporarily at least—in a man, aged fifty-one years, by daily exposure to the *x*-rays as frequently as practicable from December 1, 1903, to March 29, 1904. The sittings were of fifteen

minutes' duration for the first two months and ten minutes only during the last month. Intervals between the treatment were rendered necessary by inflammatory reaction in the skin of the neck, through which the rays were passed on alternate sides during the first few weeks, and subsequently on the diseased side only.

Reference is made to a case similarly treated with equal success by W. SCHEPPEGRELL, of New Orleans (*ibid.*, 1903, No. 11), and another by DR. MASSEI, of Nice (*ibid.*, No. 10, 1904). In none of these cases had there been any histological examinations to determine the nature of the growth.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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Studies on the Pathology and on the Etiology of Variola and Vaccinia.—From the Sears Pathological Laboratory, the Harvard Medical School; *Journal of Medical Research*, 1904, vol. xi., No. 1.

Councilman, Magrath, and Brinckerhoff describe the pathological anatomy and histology of variola, and find that the lesions of variola may be grouped under three heads: (*a*) Those which are specific and due to the presence of a parasite peculiar to the disease. (*b*) Lesions which are associated with the above, but are of indeterminate specificity. (*c*) Lesions which are caused by accessory etiological factors, among which the most important are bacteria.

(*a*) The specific lesion of variola is a focal degeneration of the stratified epithelium of the skin and of the mucous membranes of the soft palate, the pharynx, and the œsophagus. The typical lesion is best seen in the skin. It makes its appearance on about the fourth day of the disease, and, passing through progressive stages, reaches its climax of development on the eighth or ninth day. It begins with degeneration of the cells of the lower layers of the epidermis and is accompanied by exudation, at first serous, but later more or less cellular. The final stage produces the filled-out pustule. This development may take place wholly within the epidermis, or the corium may form the base of the pustule. The subsidence and repair of the lesion is accompanied by the removal of the fluid portion of the exudate by absorption, by drying, and by the regeneration of the epidermis. During this process the mass of degenerated epithelial cells, leukocytes, debris, etc., is exfoliated. Associated with the lesions of the epidermis may occur œdema, cellular infiltration, and hemorrhage of the corium. The changes in the mucous membranes are like those in the skin, except that in the absence of the horny layer the degenerated epithelial cells are cast off and the vesicle is rarely seen; the pustule never.

Within these lesions of the skin and mucous membranes is found the parasite peculiar to variola. In its younger stages or cytoplasmic form it is found within the protoplasm of the epithelial cells, usually in the earliest lesions. The intranuclear form of the parasite is present for the most part in the more advanced lesions. Variations from that form of the disease which presents typical specific lesions are determined by differences in the character and extent of the lesion.

(*b*) The associated lesions consist of a proliferation of cells in the hæmopoietic organs, in the formation of mononuclear basophilic cells in the spleen, lymph nodes, and bone-marrow, and of phagocytic endothelioid cells in the lymph nodes and marrow. Focal and interstitial infiltrations of mononuclear basophilic cells are found in the testicle, liver, kidney, and adrenal glands. In the testicle are seen anæmic focal necroses, lesions which seem to be specific. Degenerations, focal in character, are found in the blood-forming cells of the bone-marrow. Diffuse toxic degenerations occur in the liver, kidney, adrenal glands, and testicle. The paucity of polynuclear leukocytes in the specific lesions, and the focal degenerations of the bone-marrow are strikingly peculiar to the disease.

(*c*) As a result of the action of pyogenic bacteria, acute degenerations are found in the parenchymatous organs. Boils, erysipelas, and cellulitis are common sequelæ, while pharyngitis and consequent bronchopneumonia are common in severe cases, often resulting in death. Lobar pneumonia, abscess, and gangrene of the lung occasionally occur, while endocarditis, pleuritis, and empyema are relatively rare. Acute lymph noditis is present in all severe cases.

In an elaborate article Calkins describes the organism discovered by Councilman and his coworkers in the specific skin lesion of variola, and discusses its life-cycle from a morphological study. The earliest stage is the extranuclear, a cycle which exactly corresponds to the observed life-cycle of the cytorrhyctes vaccinæ. The organism is first seen in the cytoplasm of the cells of the skin as spherical homogeneous minute bodies, which are interpreted as gemmules coming from sporulation ("multiple reproduction") of the organisms of primary infection. These bodies are seen to grow, first showing vacuolization in the centre and the occasional appearance of a minute central dot. Spore-forming material, for which Calkins suggests the name "protogonoplasm," is seen to be differentiated from the rest of the organism and becomes distributed throughout the body of the organism in the form of minute spherical granules. These granules become more and more definite in the later stages of growth, and are finally liberated by disintegration of the framework of the organism, which remains as a residual structure in the host cell.

The collections of protogonoplasm or gemmules thus liberated may either infect other cells as cytoplasmic organisms, or, ultimately, may become germ cells within the nucleus. In the latter case they develop into structures which the author regards as gametocytes or sexual forms, the products of which probably conjugate, as there are intranuclear amœboid bodies which are considered as fertilized female gametocytes or zygotes. This form is the first one seen to resemble a typical cell. It becomes the pan-sporoblast and the mother cell of the primary sporoblast.

Only one sporoblast is formed by the adult intranuclear parasite of

variola. The protogonoplasm which might be called the nucleus becomes distributed by fragmentation throughout the substance of the organism, the nuclear membrane of the host cell disintegrates, and these granules of protogonoplasm escape as spores apparently by the death and disintegration of the parent organism. The spores which are liberated from the pan-sporoblast are very minute, and are characterized by the presence of a vacuole which occupies the greater part of its mass, and a thickened portion at one side. Those which find their way into the nuclei of other host cells form secondary sporoblasts by a process differing from the pan-sporoblast formation. The thickened portion at one side of the spore ultimately extends around the vacuole or vesicle, and minute vacuoles appear in it. The sporoblast becomes a mass of vesicles which determine the seats of the formation of spores that are apparently identical with those formed by the primary sporoblasts.

This secondary sporoblast becomes free in the cytoplasm of the host cell by disintegration of the nucleus. Later the cytoplasm also disintegrates, leaving the sporoblast in the more or less broken-down substance at the bottom of the pustule.

There is a characteristic sporoblast residuum after both primary and secondary sporulation.

In a study "On the Occurrence of Cytocytes Variolæ (Guarnieri) in the Skin of the Monkey Inoculated with Variola Virus," Magrath and Brinckerhoff find skin lesions comparable to those in variola vera of man. In the epithelial cells of these lesions certain structures are seen which are identical in form and in staining reaction with those in the lesions of smallpox in man. The entire developmental cycle of the parasite can be followed through both the cytoplasmic and intranuclear stages.

To determine the etiology and pathology of vaccinia, experiments were made by Tyzzer upon rabbits, calves, sheep, and adult cows. Inoculations were made in the skin, mucous membrane, and particularly the corneæ. The work was extensively controlled by inoculations of pyogenic bacteria, diphtheria toxins, and many other substances. Successful vaccinations were obtained with vaccine lymph, active vaccine virus, and human variola virus. Inoculation of the blood from smallpox patients gave uniformly negative results. In all the lesions produced by these experimental vaccinations, either from vaccine virus or human variola virus, it is possible to demonstrate cell inclusions which are specific to vaccinia and which possess a definite and constant morphology. The vaccine bodies, first claimed by Guarnieri to be intracellular parasites, are closely related in their various types, and form a series which bears a definite relation to the development of the pathological process in which they are found. The epithelial cells are first invaded by small forms in which it is difficult to distinguish structure. Becoming located near the nucleus they grow larger, and it is then possible to make out a reticular protoplasm in which is a clear spot containing a mass of basic-staining material. It seems probable that the clear spot is a nucleus, and that the granule represents the chromatin of the organism. This chromatin mass later becomes irregular and eventually divides into a number of small granules, which constitute the small infecting forms of the organism. The above series of changes was never seen in any of the epithelial cells of the lesions

produced by the control experiments, and the author is convinced that the vaccine body is an organism and represents the etiological agent in vaccinia.

Brinckerhoff and Bancroft, from their studies on the leukocyte reaction in variola, conclude that a greater or less degree of leukocytosis occurs in all cases. The typical case of severe variola which recovers without complications presents at the beginning of the eruption a normal to subnormal count, which increases with the development of the cutaneous lesions, then suffers a slight decline, rises again during the latter stages of eruption, and, finally, falls to normal during convalescence. In fatal cases the count may be high during the early stages of eruption, but from then until death steadily falls. On the other hand, in mild cases there may be no rise above the upper limit of normal, or a gradually increasing leukocytosis, reaching its acme after the skin lesions have passed their active stage. Both primary and secondary types show a marked hyperleukocytosis. The leukocyte picture is characterized by an increase in the mononuclear cell types, though the minor variations in the leukocyte curves are dependent upon fluctuations in the absolute number of polymorphonuclear neutrophiles.

Southord finds that in variola the nervous system does not present any very specific lesions. Sections of the brain and cord from cases of variola in man and of variola inoculata in the ape stained by Nissl's, Weigert's, and the Marchi methods showed no characteristic degenerations, and the only finding in the nervous system which could be regarded as an essential part of variola is the hemorrhagic tendency sometimes seen in the cortex and cord in variola hæmorrhagica.

From a clinical and experimental study of the bacteriolytic complement of the blood serum in variola, Thompson concludes that in the early stages there is a diminution in the complement of the blood, but a rapid return to normal is seen in cases unattended with secondary infection. Cases in which there is a secondary infection show a failure of the complement-content of the blood to return to normal, while a progressive continued diminution in the complement-content is seen when the case is complicated by a terminal septicæmia. It therefore seems probable that the specific contagion of variola causes a diminution of the bacteriolytic complement of the blood. If the patient is capable of resisting the contagion the complement rises, but if the infection is particularly severe, or if for some other reason the complement does not rise, the individual, as he enters the pustular stage, immediately becomes susceptible to a secondary infection by the streptococci which abound in the later skin lesions.

Macacus and Rhoesus monkeys were found by Magrath and Brinckerhoff to be susceptible to variola inoculata, the disease pursuing a typical course with more or less constitutional reaction and fever. There are lesions of the skin consisting ordinarily of a primary pock at the site of inoculation, sometimes with local secondary lesions, and less commonly with associated general exanthem. The leukocytic equilibrium is disturbed, there being a polymorphonuclear, followed by a mononuclear, leukocytosis. The disease produced in the monkey by inoculation with variola virus is not identical with variola vera in man, but agrees with variola inoculata in man.

Within the epithelial cells of the epidermis and of the hair follicles at the site of the lesions cytorrhyctes variolæ may be seen. Successful

inoculation with variola was found to confer immunity to subsequent inoculation with both variola and vaccinia. Different strains of virus from different epidemics exhibited different degrees of virulence for the monkey, those coming from epidemics of clinically severe variola producing more severe lesions and being more likely to be followed by a general eruption.

In one case the variola virus was transferred from man to monkey, from the monkey to the rabbit's cornea through four generations, and when thus inoculated on a monkey produced a protopustule followed by a general exanthem.

T. R. Bancroft reports the clinical observations on variola, his paper being based upon the study of about 1200 cases. The symptoms of onset, the eruption and temperature are discussed at some length, and the disorders of the various systems and complications of variola vera are described. The abortive forms, including that which is generally designated "varioloid," are briefly discussed.

The second interesting class, variola sine eruptione, of which 12 cases were seen in physicians and attendants, consists of an indefinite illness marked by headache, pain in the back, fever, and nausea. It is a symptom-complex which appears twelve days after exposure to variola vera, and which usually lasts about three days. After the subsidence of the fever a few definite pocks occasionally appear. The author shows sufficient evidence to warrant the belief that true smallpox may arise from contact with such patients.

The very fatal variola hæmorrhagica is divided into variola pustulosa hæmorrhagica and purpura variola, depending upon whether the hemorrhagic manifestations appear after or before the pocks. The peculiar features of these forms are discussed.

Two forms of rashes were seen in the cases of smallpox, the initial and the secondary rashes. Two forms of initial rashes were observed: an erythema, which usually appeared on the second day of the disease, and which varied from a mere flushing of the face to a general erythema, occurred in 16 cases; the petechial form, which seldom occurred unassociated with erythema, was seen in 10 cases.

Of the secondary rashes, also, two forms were seen; a fine, red, maculopapular eruption and a mottled erythema. The first mentioned usually appeared soon after the climax of light cases of variola, while the second form occurred in the course of the more severe types, usually about the thirteenth day, and was preceded by constitutional symptoms. This mottled erythema was at first regarded as a secondary scarlet fever, but further evidence was against this assumption.

The Changes Produced in the Hæmolymph Glands of the Sheep and Goat by Splenectomy.—WARTHIN, in the contributions to *Medical Research*, dedicated to Victor C. Vaughan, June, 1903, reports a series of experiments upon sheep and goats in which the changes produced in the hæmolymph glands by splenectomy were studied. Eight animals were used, and autopsies were performed from twenty-four hours to five months after the removal of the spleen. The author finds that after total splenectomy in the sheep there is no evidence of regeneration of the primitive spleen or of the new-formation of splenic tissue. According to his observations the structural changes following splenectomy are: hyperplasia of existing lymphoid tissues, transformation

of hæmolymph nodes into ordinary lymphatic glands, and a new-formation of hæmolymph nodes out of the lobules of the fat tissue, and a later proliferation of the red marrow. He finds no evidence of the formation of red blood cells in the lymph nodes after splenectomy. The function of hæmolysis, however, is taken up first by the hæmolymph node, and later by the ordinary lymphatic gland.

The author considers that the hæmolytic function of the hæmolymph glands and hyperplastic lymph glands exceeds that of the primitive spleen, causing an excessive destruction of red cells. The resulting anæmia is later compensated for by an increased activity on the part of the bone-marrow. It would appear, therefore, that the removal of the spleen leads to an increased production or retention of some hæmolytic agent usually disposed of by the spleen. The effect of this hæmolytic agent is either to stimulate the phagocytes in the hæmolymph glands to increased activity or to change the red cells so that they are more easily destroyed by these phagocytes.

The greater number of eosinophiles found in the glands showing great destruction of red cells, the author considers to point to some relationship between these cells and hæmolysis.

The work confirms the observations of several former experimenters, all of whom, however, put different interpretations upon the appearances which they found to follow splenectomy.

Thirteen photomicrographs show the principal changes described.

A Comparison of the Animal Reactions of the Trypanosomes of Uganda and Congo Free State Sleeping Sickness with Those of Trypanosoma Gambiense (Dutton).—THOMAS and LINTON (*Lancet*, 1904, i., 1337) have found from inoculations of these three types of organisms in mice, guinea-pigs, rabbits, cats, puppies, goats, donkeys, and monkeys that the trypanosomes found in (*a*) cerebrospinal fluid of Uganda sleeping sickness, (*b*) cerebrospinal fluid of Congo Free State sleeping sickness, (*c*) blood of Uganda trypanosome fever cases, and (*d*) blood of Congo Free State fever cases, are all identical in animal reactions and morphology with trypanosome gambiense (Dutton). They believe the specific name gambiense must for the future include the trypanosomes from the above-mentioned sources. Further, there seemed to be no immunity against infection, and no transmission of immunity to offspring. Animals which seemed to have recovered showed months later parasites once more.

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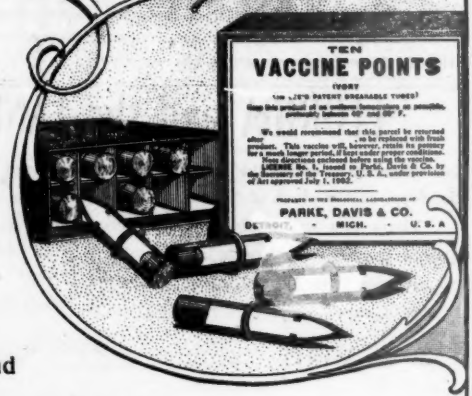

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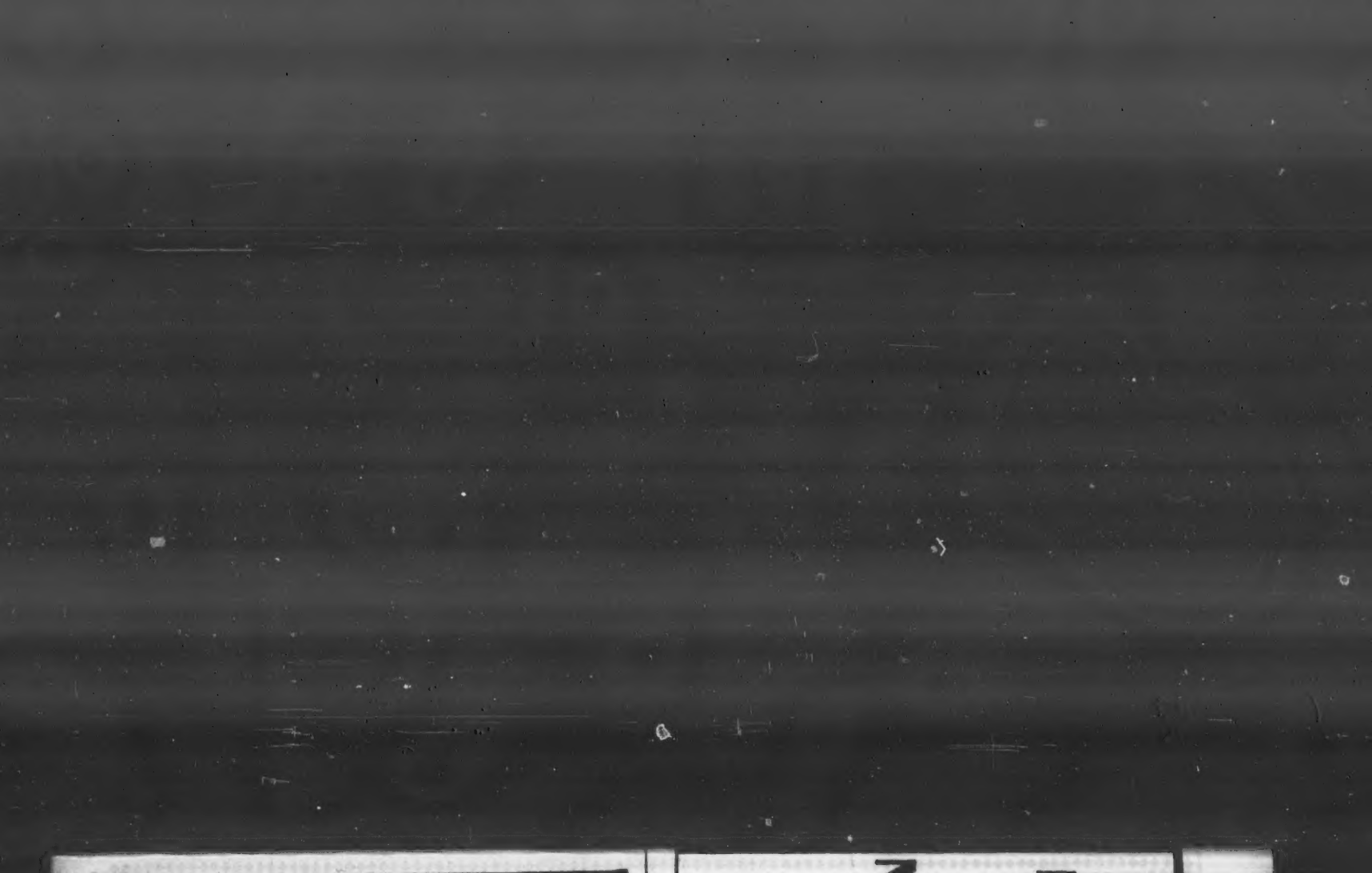

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